“A STUDY TO EVALUATE THE BONE MARROW CHANGES IN GERIATRIC PATIENTS WITH ANEMIA”

DISSERTATION SUBMITTED FOR

M.D. DEGREE EXAMINATION

BRANCH III PATHOLOGY

of

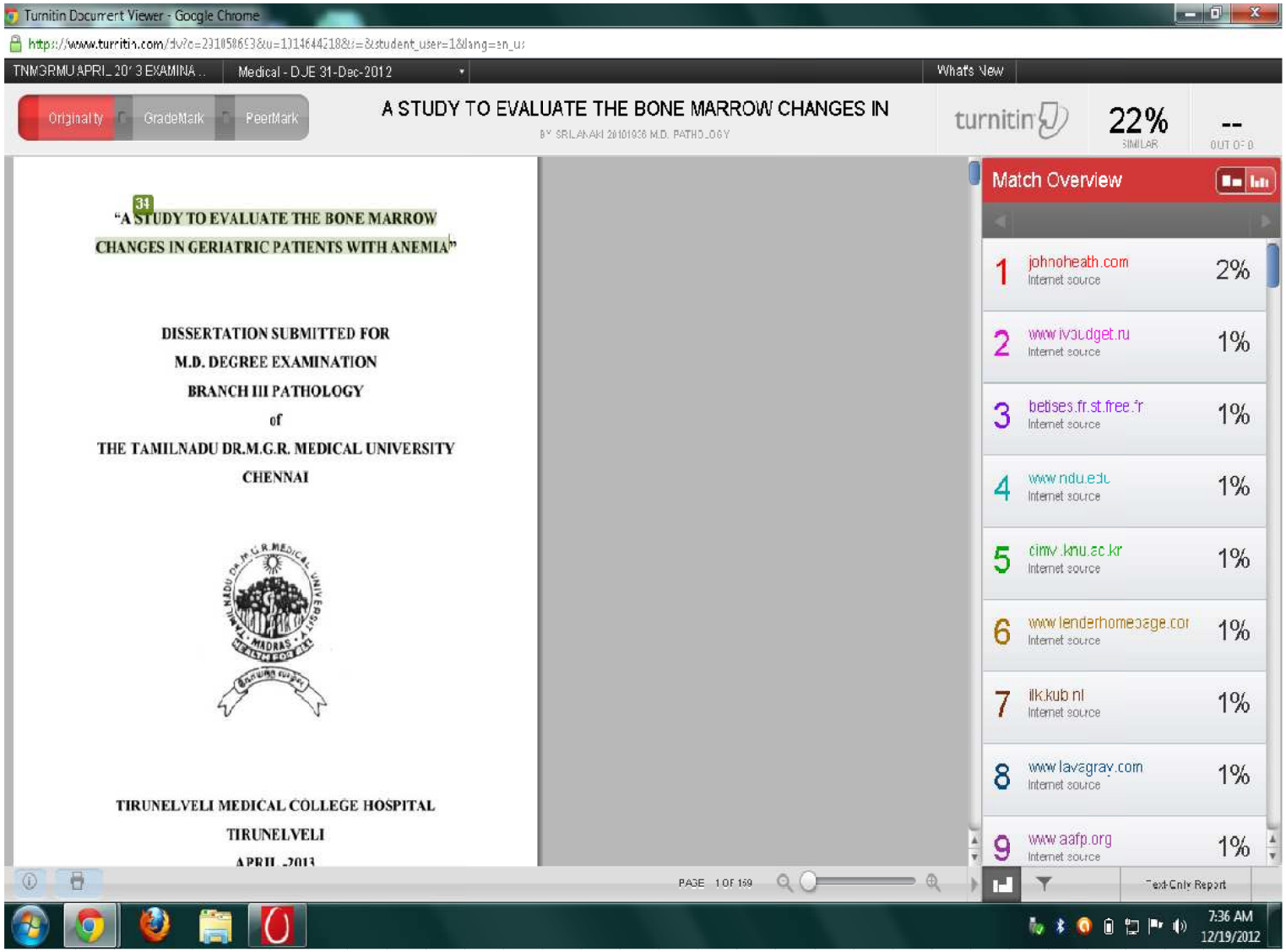
THE TAMILNADU DR.M.G.R. MEDICAL UNIVERSITY

CHENNAI

TIRUNELVELI MEDICAL COLLEGE HOSPITAL

TIRUNELVELI

APRIL -2013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This is to certify that the Dissertation **"A STUDY TO EVALUATE THE BONE MARROW CHANGES IN GERIATRIC PATIENTS WITH ANEMIA"** presented herein by **DR. SRI JANAKI** is an original work done in the Department of Pathology, Tirunelveli Medical College Hospital, Tirunelveli for the award of Degree of M.D. (Branch III) Pathology under my guidance and supervision during the academic period of 2010 - 2013.

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I hereby certify that this work embodied in the dissertation entitled **"A STUDY TO EVALUATE THE BONE MARROW CHANGES IN GERIATRIC PATIENTS WITH ANEMIA"** is a record of work done by **DR. SRI JANAKI** in the Department of Pathology, Tirunelveli Medical College, Tirunelveli, during her postgraduate degree course in the period 2010-2013. This work has not formed the basis for any previous award of any degree.

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I solemnly declare that the dissertation titled **"A STUDY TO EVALUATE THE BONE MARROW CHANGES IN GERIATRIC PATIENTS WITH ANEMIA"** is done by me at Tirunelveli Medical College Hospital, Tirunelveli.

The dissertation is submitted to The Tamilnadu Dr. M.G.R.Medical University towards the partial fulfilment of requirements for the award of M.D. Degree (Branch III) in Pathology.

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ACKNOWLEDGMENT

I thank the Dean and Vice Principal of the Tirunelveli Medical College, Tirunelveli for the kind permissions they had accorded for the study.

I express my sincere gratitude to Dr. Sithy Athiya Munavarah, Professor and Head of the Department of Pathology, Tirunelveli Medical College, Tirunelveli for her valuable guidance and appreciation in the conduct of this study.

I express my gratitude to my guide Dr.K.Shantaraman, Professor of Pathology, Tirunelveli Medical College, Tirunelveli for his resolute guidance and meticulous supervision through the course of this work and preparation of manuscripts.

I express my sincere gratitude to Dr. K.Swaminathan, Dr.S.Vallimanalan, Dr.Suresh Durai Dr.Arasi Rajesh, Professors in the Department of Pathology, Tirunelveli Medical College, Tirunelveli for their valuable guidance and appreciation in the conduct of this study.

I express my sincere thanks to all Professors, Assistant Professors and Residents in the Clinical Departments and the Assistant Professors, Tutors and Staff in the Department of Pathology for their support.

I thank all my fellow post graduates for their kindness, camaraderie and support.

My sincere gratitude to all my study subjects whose cooperation has contributed to this study.

Date:

Place: Tirunelveli

ABBREVATIONS

AOCD	:	Anemia Of Chronic Disease
APD	:	Acid Peptic Disease
AML	:	Acute Myeloid Leukemia
CML	:	Chronic Myeloid Leukemia
CAHD	:	Coronary Artery Heart Disease
DM	:	Diabetes Mellitus
HB	:	Hemoglobin
HT	:	Hypertension
H/O	:	History Of
IDA	:	Iron Deficiency Anemia
MCHC	:	Microcytic Hypochromic
MDS	:	Myelodysplastic Syndrome
REH	:	Reactive Erythroid Hyperplasia
UGI	:	Upper Gastro Intestinal
WBC	:	White Blood Cell
WHO	:	World Health Organisation

CONTENTS

INTRODUCTION

Aging is not a cause of anemia; but is seen as a predisposing factor to anemia. Anemia is a common hematological problem in the geriatric age group and its incidence increases with increasing age[1]. Studies have demonstrated that anemia in old age have unique causes, adverse effects and functional outcome[2]. The prevalence of anemia in the general population has been shown to range from 8 to 44%[3]. The third National Health and Nutrition Examination Survey (NHANES-III) of WHO study revealed prevalence of anemia as 11% of men and 10.2% of women aged 65 years and older[4]. The etiology is multi-factorial, the most common causes being Anemia of Chronic disease, Nutritional deficiencies followed by Unexplained anemia[4]. Myelo-dysplastic Syndrome (MDS) is more common in older adults and accounts for a significant number of unexplained anemias in elderly[4]. The median age at which MDS is diagnosed is between 60-75 years. To understand the cause and effect of anemia on the old person, it is essential to look into the bone marrow for specific changes, for example the diagnosis, classification and prognostication of MDS is, based on the peripheral blood film and Bone Marrow morphology of the patient[5]. Hence Bone Marrow Morphology remains the cornerstone and most often the gold standard of disease diagnosis and is an important tool that complements cytogenetic finings for prognostic discrimination[6]. Our present study was designed as a prospective descriptive study of the Bone Marrow morphology in Geriatric patients with Anemia.

AIMS AND OBJECTIVES

We aim

1. To evaluate the clinical presenting features and the basic, hematological parameters in Geriatric patients with Anemia.
2. To evaluate the morphological alterations of Bone marrow aspirates in these Geriatric patients with Anemia.
3. To correlate these morphological alterations of Bone marrow aspirates with the clinical and basic hematological parameters in these patients.

REVIEW OF LITERATURE

I. The Elder

Geriatrics or **geriatric medicine** focuses on health care of elderly people. The term *geriatrics* comes from the Greek word *geron* meaning "old man" and *iatros* meaning "healer". The age sixty five is considered the mean geriatric age and according to Zauber & Zauber(1987), the older individuals can generally be classified into three age categories– the young old, age sixty five to seventy four years, the old- old, age seventy four to eighty four and the very old, age eighty five and older[7]. The number and proportion of elderly persons is increasing worldwide and this contributes to the global growing population. Currently there are nearly five hundred million (7%) adults sixty five years and older throughout the world and by 2030, the older population will double to one billion (12%) [8]. Of the total world population of 6,464,750, nearly 5,253,484 live in the lesser developed regions, and about two thirds of all geriatric people are living in these developing regions. By the year 2025, this number is projected to rise further[9]. This shifting demographic trend demands the health care system to get equipped accordingly to serve the present and the future need of the community. Although advanced age is considered a risk factor for many diseases it does not equate age, disease and disabilities. The decline in physiological reserve in organs makes the elderly more prone for diseases and have more complications from mild

problems. Over the past decade, Anemia has grown as an important risk factor and is associated with an array of undesirable outcomes in elder adults, including hospitalization, disability and mortality. Anemia is an insidious disease and is associated with a wide range of negative health conditions in the elderly. The continuum of conditions range from a simple decrease in physical performance and muscular activity, to severe complications that lead to hospitalization and mortality (Denny SD et al., 2006; Penninx BW et al.,2003 and 2006)[10,11] Anemia can have significantly more severe complications in the older individuals than in the young and are bound to impede the quality of life. The prevalence of anemia range from eight to forty four percent with a significant increase in men who are 85 years or older. The third National Health and Nutrition Examination Survey (NHANES-III) of WHO study estimated the prevalence of anemia as eleven percent in men and 10.2% in women aged sixty five years and older. Guralnik JM et al. (2004) estimated that the prevalence of anemia is 10.6% in people who were 65 years and older. This study, based on the data from Phases 1 and 2 of the NHANES III survey, showed that in individuals over the age of 85, the prevalence of anemia rose to 26.1% for males and 20.1% for females. It also revealed that anemia has an impact on mortality in geriatric patients with other co-existing disease states, which is of greater significance. Although anemia

is common in the elderly, a low hemoglobin level cannot simply be dismissed as a normal part of aging.

II. Normal hematopoiesis

The process of Hematopoiesis begins in the yolk sac at third week of intra uterine life. From third month onwards, Hematopoiesis starts in Liver which continues up to sixth month. Bone marrow starts taking over the function from fourth month onwards and from then on it is almost the only source of blood cells at and after birth[12].

Bone marrow is made up of (a) cellular elements of hemopoietic stem cells, proliferating and differentiating progenitors and morphologically recognizable precursors and (b) stroma. There is a critical interaction between stromal cells and the hemopoietic progenitor cells[12]. Islands of Erythroblasts are seen in central parts of the marrow in close proximity to sinusoids. Myeloblasts are frequently found adjacent to the trabecular surfaces. Megakaryocytes are normally present in the central parts of the bone marrow amongst the erythroid and myeloid precursors. Monocytic precursors are usually not recognizable in the marrow sections. Lymphocytes are seen interstitially and also around arterial vessels, in the central parts of the marrow.

The process of hematopoiesis begins with the production of blood cells from hematopoietic stem cells (HSC) which have extensive potential of proliferation to produce more stem cells (self-renewal) and

differentiate in to progenitor cells. Progenitor cells (lineage committed cells) are committed to one lineage, i.e., myeloid or lymphoid. Their proliferative potential is limited as compared to HSCs. Late progenitor cells (committed progenitor cells) possess receptors for various cytokines. Late progenitor cells differentiate into morphologically recognizable maturing cells like proerythroblasts, early normoblasts etc. As these cells mature, proliferative potential decreases and finally mature white cells, red cells and platelets are produced.

Stem cells in the marrow are surrounded by non-hemopoietic cells and proliferating and differentiating precursors. Non- hemopoietic cells like stromal cells, macrophages, fibroblasts, endothelial cells, fat cells etc form the microenvironment of marrow which controls the self renewal and differentiation of HSCs.

Hemopoietic growth factors (cytokines) are hormone like inducers of proliferation and differentiation of HSCs. These cytokines act on progenitor cells through surface receptors and their action is lineage specific[12].

III. Hematopoiesis in the Elder

The Aging process is associated with a decline in function of multiple organ systems, such as cardiovascular system, renal system, pulmonary, musculo-skeletal system and notably the hematopoietic process. As age advances, most cells lose their ability to divide, however,

the Bone Marrow and gastro intestinal mucosal cells retain their mitotic activity. In infancy, the Marrow is estimated to occupy 80% to 100% of the medullary spaces of long bones and a few flat bones, which gradually shrinks with advancing age decreasing to 50% by 30 years of age, and these further declines to 30% by 65 years of age[13].

Erythrocytes:

Anemia is frequently found in elderly individuals. The stimulating effects of androgens on erythropoiesis results in a higher hemoglobin levels in men when compared to women. Unlike in young and middle aged persons, both elderly men and women have an equal chance of developing anemia because of a decrease in androgen levels in elderly males (Allan RN & Alexander MK; 1965) [14].

Leukocytes:

The total leukocyte count or WBC differential count remains almost normal in healthy elderly persons with no underlying pathologic conditions, when compared with middle aged individuals. [14].

Lymphocytes:

Age allied defects in lympho cytogenesis such as immune senescence and other factors, affects cellular and hum oral immunity. The thymus disappears by early middle age, and from then on the adults depend on the T cell responses in other secondary tissues. The number of naïve T cells decreases as age advances, thus increasing the dependency

on memory T cells. In the elderly, an impaired responsiveness of Tcells to mitogens and antigens is present as a result of a decreased expression of co-stimulator CD28. The function of B lymphocyte depends on interaction with T cells. When T cell inadequacies occur, there may be a decreased ability to generate an antibody response[14].

Platelets:

The platelet count is not altered significantly with age. However, the function of platelets may be altered due to decreased platelet membrane protein kinase C activity and increased levels of ß-thromboglobulin[14].

IV. Anemia in the Elder

Anemia, is not a diagnosis in itself, but is an objective sign of a deeper disease. Anemia is defined as a condition in which the concentration of hemoglobin in peripheral blood is below the level that is normal for the level for that age and sex of the individual[15]. Functionally, it is defined as an insufficient RBC mass to adequately deliver oxygen to the peripheral tissues[16]. The normal limit of hemoglobin concentration varies among different organizations, countries and laboratories. In addition, many other factors can influence a healthy person's hemoglobin level, including smoking status, altitude of residence, ethnic background, and physiologic fluctuations of plasma volume[17]. Because clinical laboratories do not adjust for these factors, interpretation of hemoglobin

test results is the responsibility of the clinicians. The World Health Organization(WHO) criteria of anemia is based on a hemoglobin concentration lower than 120g/L(12.0G/dl) in women and 130g/L(13.0g/dl) in men[18]. The interpretation of hematologic data in the context of old age is particularly a matter of concern nowadays. This could partly be attributed due to the remarkable heterogeneity of the processes related to senescence and partly due to the intricacies in sorting out the effects of age per se from the effects of the occult disease processes that go together with aging. This results in an ambiguous situation wherein it is unclear whether the low hemoglobin levels observed is primarily due to age or an underlying disease. Many elderly people maintain normal blood counts in spite of low hemoglobin levels. Many variables are postulated that contribute to anemia in the elderly such as (1) decline in physical activity, (2) altered cardiovascular function, (3) decreased bone marrow function and (4) chronic inflammatory disorders[19]. This process of an undercurrent anemia possesses a higher mortality risk of anemic elderly than in non anemic elderly.

Table 1: Grading of the severity of anemia[20]

Grade of Severity	Hemoglobin Level
Within Normal Limits	>12gms/dl
Mild	10 - 11.9gms/dl
Moderate	7.1 - 9.9gms/dl
Severe	< 7.0gms/dl

V. Classification of anemia[21]

I. Physiologic Classification

II. MorphologicClassification

I) Physiologic Classification

A) Blood loss

B) Increased rate of destruction (hemolytic anemias)

1) INTRINSIC (intracorpuscular) abnormalities of red cells

A) Hereditary

I. Red cell membrane disorders:-

- Disorders of membrane cytoskeleton
- Disorders of lipid synthesis

ii. Red cell enzyme deficiencies

- Glycolytic enzymes
- Enzymes of hexosemonophosphate shunt.

iii. Disorders of hemoglobin synthesis

b) Acquired

- Membrane defect

2) EXTRINSIC (Extra corpuscular) abnormalities

a) Antibody mediated

b) Mechanical trauma to red cells

c) Infections: malaria, hookworm

d) Chemical injury**:** lead poisoning

e) Sequestration in mononuclear phagocyte system: hypersplenism

C) Impaired red cell production

(i) Disturbance of proliferation and differentiation of stem cells:

(ii) Disturbance of proliferation and maturation of erythroblasts

MORPHOLOGICAL CLASSIFICATION

I. Normocytic Anemias

II. Microcytic Anemias

III. Macrocytic Anemias

VI. Etiology of Anemia in Elder

The principal causes of anemia in geriatric persons were evaluated in the Third National Health and Nutritional Examination Survey (NHANES III) and are as follows[4]:

1. One third were related to Blood Loss / Nutrition deficiency - Iron deficiency and iron with folate and/or B12 deficiency , Folate and/or B12 deficiencies.
2. One third due to Chronic Renal disease, Anemia of Chronic disease or both.
3. Remaining one third was Unexplained Anemia.

In another study, the Stanford study, Unexplained anemia accounted for 35%, hematologic malignancy accounted for up to 22%, Iron deficiency anemia 12%, Anemia of Inflammation 6%, Renal insufficiency 4% and others 10%[27]. In addition Drug induced anemia secondary to various medications such as Nonsteroidal Anti-inflammatory drugs, anti-platelet medications, steroids, etc are well known to occur in elderly. One of the challenges in identifying the etiology of anemia in elderly patients is the diversity of potentially contributing factors (eg, alcohol use and medications). Thus, anemia in older persons is multifactorial, and evaluation of low hemoglobin may be more challenging than in adolescents or young adults.

VII. Pathophysiology of Anemia in Elder

1. Anemia of Inflammation:

Anemia of chronic disease, or Anemia of chronic Inflammation, is the most common form of anemia in the elderly. The anemia that is observed in patients with chronic infections, inflammatory disorders, or malignant diseases that persist for more than one to two months is called Anemia of Chronic Disease (AOCD)[22]. Elis A et al (1996)[23] have shown that most common anemia in elderly is Normocytic Normochromic type. The most common conditions associated with AOCD are rheumatologic disorders, particularly, Rheumatoid Arthritis, Tuberculosis, Pelvic Inflammatory Disease(PID), AIDS/HIV, Systemic Inflammatory Response Syndrome(SIRS), Chronic Osteomyelitis, Inflammatory Bowel Disease(IBD), Hodgkin's and Non-Hodgkin's lymohomas and Cancers of various organs. Classically, it is characterized by the occurrence of hypoferrenemia in the presence of normal or abundant reticulo-endothelial iron stores. On a biochemical basis, it is characterized by a low serum iron level, low to normal transferrin level and low iron binding capacity with an elevated serum ferritin.

The etiology is multifactorial**.** Three principal mechanisms are involved in the pathogenesis of AOCD[24]. These are (1) a shortened erythrocyte survival, due to a potential extra corpuscular defect (2) impaired marrow response due to inhibitory effect of cytokines on

erythropoiesis, and (3) disturbance in iron metabolism, in which iron in the bone marrow macrophages is not available for erythropoiesis. Cytokines that are frequently implicated in the pathogenesis of AOCD are IL-1, IL-6, TNF, and the Interferons, The serum or plasma concentrations of these cytokines are increased in patients with disorders associated with AOCD. Recent studies reveal that the key factor in the pathogenesis of AOCD is the antimicrobial peptide Hepcidin[25]. It is produced by the hepatocytes and is a principal regulator of iron metabolism. It has been shown to hamper iron absorption from the intestine and to arrest release of iron from enterocytes, hepatocytes and macrophages. Elevated levels of hepcidin have been demonstrated in patients with AI. Various complex mechanisms involving a number of inflammatory-mediated cellular pathways have been implicated in the regulation of hepcidin synthesis. Hepcidin is an acute phase reactant and its synthesis is potently induced by IL-6. The In CHIANTI study[26] or "Invecchiare in Chianti" study carried out recently analyzed the association of hepcidin levels in urine, proinflammatory markers, and anemia. In this study it was observed that although there was an association of C-reactive protein and IL-6 with anemia and reduced iron levels, no association was found with higher urinary hepcidin levels. Hence from this study it was postulated that an increase in hepcidin synthesis occurs only in presence of blatant inflammation. Also in AI, elevated lactoferrin levels, interferes with

normal iron cycling and contributes to increased iron stores. **Laboratory Features:** Characteristically, serum iron concentration is decreased, total iron-binding capacity (or serum transferrin concentration) is reduced, and transferrin saturation is subnormal though not to the same degree as in iron deficiency. Patients with Anemia of Chronic inflammation have mild to moderate anemia, and a variable hypochromia. The degree of anemia tends to correlate in severity with the underlying disease, and rarely may it progress to a much higher degree. In AOCD, the RBCs are usually normochromic and normocytic, but in about one third of patients with AI, they may be microcytic. Hypochromia is more common and precedes microcytosis, unlike in iron deficiency, where hypochromia typically follows microcytosis. This feature often helps in distinguishing Iron deficiency anemia from AOCD. A mild anisocytosis and poikilocytosis may be detected, but these changes tend to be less prominent than in iron-deficient persons. There is no increase in reticulocyte count. Bone marrow is normocellular or mildly hypercellular with a normoblastic hematopoiesis. The reticulo endothelial iron content is increased. Normal sideroblasts contain one to four tiny blue inclusion bodies in Prussian blue iron staining. In AI, the number of Sideroblasts is reduced to 5 to 20% of the total quantity of Normoblasts (normal, 30 to 50%). In Bone marrow aspirates stained for iron, in iron deficiency

anemia, both the macrophage iron and the number of sideroblasts are reduced.

AOCD has to be differentiated from Anemia of Renal disease, chronic blood loss, drug induced hemolysis or Marrow suppression, Thalassemias, Endocrine disorders such as testicular failure, hypothyroidism and hyperthyroidism. These conditions need to be ruled out before assigning a patient with the diagnosis of AOCD. Treatment of the underlying disease usually resolves the anemia. Other treatment modalities include erythropoietin therapy and blood transfusion.

2. Anemia in Patients with Cancer:

Much of the anemia commonly observed in patients with Cancer can be attributed to the mechanisms involved in ACD; however, certain processes unique to Malignancy may also contribute. The potential mechanisms are - Erythroid precursors may be displaced from marrow by metastatic tumor, tumor-induced fibrosis, or tumor-associated marrow necrosis. The treatment of Cancer can also produce or exacerbate anemia by a variety of mechanisms, including impaired erythropoietin production and cytotoxic effects of therapy on erythroid progenitors[2].

3. Anemia of Renal Disease:

Anemia of Chronic Renal Insufficiency is due to loss of interstitial cells of kidney with reduction in production of erythropoietin (EPO) in response to hypoxia. The severity of anemia increases as the renal

function is compromised. In addition anemia is further accentuated because of (a) suppression of erythropoiesis by toxic metabolic products of renal failure[30]. (b) reduce red cell survival (c) bleeding due to impaired platelet function. Progressive uremia results in plasma expansion and hemodilution. There is functional iron deficiency due to depletion of iron pool since the increased iron stores are not available for utilization by the bone marrow for erythropoiesis. Additional factors associated with chronic renal failure will also contribute to the development of anemia, and these should be regarded as complications and not fundamental components of the Anemia of Renal insufficiency itself. For instance, in the presence of infection or inflammation associated with renal disease, Anemia of chronic disease is likely to be observed. Iron deficiency anemia may develop because of hematuria, blood loss from the gastrointestinal tract or from retention of blood in the hemodialysis apparatus tubing. The Megaloblastic anemia due to folate deficiency may also occur in patients on dialysis but is rare. Certain types of renal disorders, including the Hemolytic-Uremic syndrome or Thrombotic Thrombocytopenic purpura, are associated with Microangiopathic Hemolytic Anemia. Finally, aluminum intoxication can cause Microcytic anemia in dialysis patients. The blood film usually shows normocytic normochromic anemia along with few burr cells and poikilocytes. Reticulocyte is low for the degree of anemia. The bone marrow is usually

normocellular with nearly normal erythroid elements. In chronic renal failure, iron stores are increased unless the patient has got associated iron deficiency. Bone marrow biopsy, besides showing increased iron stores, demonstrates a picture of Renal Osteodystrophy especially in cases of advanced chronic renal failure. The bony trabeculae demonstrate increased osteoblastic and osteoclastic activity with lacunar resorption of bone resulting in saw tooth irregularity of the trabecular surface. There is associated paratrabecular fibrosis. Patients respond to erythropoietin therapy[27].

4. Anemia of Liver Disease:

The term Anemia of Liver disease refers to a mild to moderate anemia associated with liver disease in the absence of any complicating factors such as Blood loss, Marrow suppression by exogenous agents, or Nutritional deficiency. This syndrome apparently results from a combination of intravascular dilution due to volume overload, shortened red cell survival, and impaired ability of the marrow to respond optimally to the anemia. In addition, some patients develop a severe hemolytic anemia associated with morphologically abnormal erythrocytes (spur cells). Alcohol abusers can develop a characteristic Sideroblastic anemia, often accompanied by impaired folate metabolism or overt folate deficiency or may have direct suppression of hematopoiesis by alcohol. Individuals with cirrhosis of any etiology are at increased risk for

hemorrhage. Blood loss occurs in 24 to 70% of patients with alcoholic cirrhosis. In liver diseases, a Normocytic or Macrocytic anemia develops[27].

5. Anemia of Endocrine Disorders:

Many Endocrine disorders may initially manifest with anemia. The common disorders that produce anemia are Hypothyroidism, Hypopituitarism, and Adrenal insufficiency. The anemia is mild to moderate and is usually not associated with symptomatology (other than that associated with the underlying endocrinopathy). However the severity of anemia may be masked by a decreased plasma volume that occurs in some disorders. The anemia is "Adaptive". and may reflect a physiologically appropriate hemoglobin concentration because the hormone deficiency often results in reduced oxygen requirements. In most of the endocrine diseases the anemia is Normocytic and Normochromic but may be Macrocytic[27].

6. Anemia of Collagen Vascular Diseases:

Anemia of Chronic disease appears in Collagen Vascular diseases such as Dermatomyositis, Polyarteritis nodosa, Rheumatoid arthritis, Systemic Lupus Erythematosus, and Temporal arteritis (including polymyalgia rheumatica). The latter is essentially a disease of geriatric patients[27]

7. Iron Deficiency Anemia:

Iron deficiency anemia produces a Microcytic Hypochromic anemia. It is the most common form of anemia prevalent in India[28]. It is the most wide spread form of nutritional anemia throughout the world[29]. Common causes of IDA include lack of dietary iron, chronic blood loss and malabsorption states. In geriatric individuals, chronic bleeding from the Gastro intestinal tract and Genitor urinary tract are major causes apart from nutritional deficiency. The gastro intestinal causes of blood loss include Peptic ulcer, Oesophageal or Gastric Varices, Hemorrhoids, NSAID intake, vascular anomalies such as Angiodysplasia and Malignancies. Other less common causes are chronic respiratory tract infections, renal, bladder and prostatic lesions etc. The pathogenesis of iron deficiency anemia involves (i) impaired hemoglobin synthesis (ii) impaired cellular proliferation (iii) diminished iron containing proteins.

Laboratory Findings:

Diagnosis is based on peripheral blood findings, bone marrow morphology and iron stores and assessment of Iron status. Low levels of serum Iron, serum Ferritin and an increased serum Iron binding capacity is noted. An increase in plasma transferrin receptor is a sensitive indicator of tissue iron deficiency. The Mean Corpuscular Volume and Mean Corpuscular Hemoglobin values are reduced in most patients, and the Mean Corpuscular Hemoglobin Concentration is reduced in long-

standing or severe disease. The Red cell Distribution Width (RDW) is increased in iron deficiency, and this often is useful in distinguishing iron deficiency from Thalassemia trait conditions in which the RDW is normal. The chief finding on blood smear is hypochromia, observed as an increase in the size of the region of central pallor. The more severe the anemia, the more severe the hypochromia and the greater the percentage of erythrocytes affected. When hypochromia is extreme, most of the red blood cells appear as mere rings. Tiny microcytes and a moderate number of poikilocytes, particularly tailed and elongated elliptical forms (pencil cells), are also found. The total white blood cell count is usually normal, but mild reduction in polymorphs may occur in long-standing cases of iron deficiency. Recent large-volume hemorrhage may cause a slight neutrophilic leukocytosis, and occasional myelocyte may be found in peripheral blood. In iron deficiency due to parasitic infestation, eosinophilia may be present. Another feature that commonly accompanies iron deficiency is an elevated platelet count. The thrombocytes are often increased to approximately twice the normal level, but the number return to normal after iron treatment. The cause of the thrombocytosis of iron deficiency is unclear.[30]

Bone Marrow Finding:

There is absent or severely reduced Macrophage iron in the marrow and also in spleen, and liver of iron-deficient subjects. Fewer than 10% of

the marrow normoblasts are Sideroblasts. In addition, the iron-deficient bone marrow is characterized by mild to moderate Erythroid hyperplasia. There may be striking nuclear distortions, resembling those found in Dyserythropoietic anemias. Karyorrhexis and nuclear budding are particularly common, but multinuclearity, nuclear fragmentation, and even intranuclear bridging may be observed[31]. The individual normoblasts appear small and may have scanty cytoplasm, often with irregular, ragged borders. When therapy is given, Erythroid hyperplasia initially increases, but as erythropoiesis is restored to normal, the cellularity of the marrow likewise becomes normal.

The differential diagnosis includes Thalassemias, AOCD and Sideroblastic anemia. The Anemia of Chronic disease may be microcytic, but it is more commonly normocytic. Iron stores are normal and clinical features of chronic disorders are usually present. Sideroblastic anemia may be microcytic, normocytic, or even macrocytic. Bone marrow demonstrates increased iron stores with ring sideroblasts.

8. Vitamin B12 / Folate Deficiency:

Vitamin B12 / Folate deficiency produce a Macrocytic anemia. Megaloblastic anemias are characterized by retarded DNA synthesis while RNA synthesis is normal resulting in an imbalanced cell growth. The Mean Corpuscular Volume increases slightly with age but it generally does not lead to significant Macrocytosis. Though serum levels

of Vitamin B12 are subnormal (10-15%), anemia due to B12 deficiency is not so common in elderly (1-2%)[32]. Common causes of cobalamin deficiency are inadequate diet, malabsorption, increased requirements as in disseminated cancer and intrinsic factor deficiency. Pernicious anemia [is] a chronic disease resulting from deficiency of intrinsic factor leading to impaired absorption. It is more common over the age of 60 years[33]. The etiopathogenesis involves damage to gastric parietal cells caused by cellular and humoral immune reactions leading to impaired secretion of hydrochloric acid, pepsin and intrinsic factor.

9. Folate Deficiency:

Like Cobalamin deficiency, Folate deficiency characteristically causes Macrocytic anemia. Simultaneous iron deficiency may mask macrocytic anemia resulting in a normocytic anemia especially in elderly persons[34]. The causes of folate deficiency are unbalanced diet; impaired absorption associated with Tropical sprue, small bowel resection and increased requirements as in Hemolytic anemias, Myeloproliferative disorders and hemodialysis. The distinction of cobalamin and folic acid deficiency is necessary as anemia resulting from cobalamin deficiency improves with folate therapy, but folic acid therapy does not revert the neurologic damage caused by cobalamin deficiency.

Other causes of megaloblastic anemia are defective folate/ vitamin B12 metabolism, Hereditary Orotic aciduria and Lesch- Nyhan syndrome.

Laboratory Diagnosis:

Hematologic findings are similar in both B12/Folate deficiency. The classic blood count findings are anemia, a high MCV and Mean Corpuscular Hemoglobin (MCH), and, in more advanced cases, thrombocytopenia and neutropenia. The reticulocyte count is low due to ineffective erythropoiesis. Also there is an increase in lactate dehydrogenase levels, unconjugated bilirubin, and low serum haptoglobin levels due to a poorly understood component of intravascular hemolysis in these patients. The characteristic features in peripheral smear are hypersegmented neutrophils and macro ovalocytes. Hypersegmentation of neutrophil nuclei is a constant feature and is defined as finding one or more neutrophils with six or more nuclear lobes or showing that at least 4 to 5% of neutrophils have five lobes. Hypersegmentation often precedes anemia, but it is not found in subclinical deficiency. Hypersegmented neutrophils are not specific for cobalamin or folate deficiency; they are found in patients receiving chemotherapeutic drugs such as 5-fluorouracil or hydroxyurea, in some patients receiving steroid therapy for Immune Thrombocytopenic Purpura , in rare patients with Myelofibrosis or Chronic Myelogenous Leukemia, and, possibly, as a benign hereditary condition. Macro ovalocytes are large cells (MCV>100fl) and are pathognomonic of Megaloblastic anemia. Other features that can be present are anisopoikilocytosis, Howell- Jolly bodies, cabot rings,

basophilic stippling, nucleated RBCs, leucopenia and thrombocytopenia[35]. *Bonemarrow Findings* The bone marrow is markedly hypercellular with erythroid hyperplasia and a reversal of myeloid erythroid ratio. The morphologic hallmark is nuclear-cytoplasmic dissociation, best appreciated in precursor cells in the bone marrow aspirate. There is a preponderance of megaloblastic changes in early and intermediate erythroblasts. Megaloblasts are characterized by a large cell size, large, a distinctive fine nuclear chromatin pattern (due to a preponderance of euchromatin). Cytoplasmic maturation and hemoglobin accumulation proceed at a normal pace, leading to nuclear to cytoplasmic asynchrony. Morphologic aberrations apparent in myeloid cells are giant metamyelocytes and band forms. Megakaryocytes display unusually large and bizarre multilobated nuclei[36].

Apart from vitamin B12/ folate deficiency, liver disease, hemolytic anemia, alcoholism, hypothyroidism, aplastic anemia, Myelodysplastic syndrome, and cytotoxic chemotherapy also produce macrocytic anemia and needs to be distinguished from B12/ folate deficiency. In these conditions, MCV rarely exceeds 120fl and the bone marrow reaction is normoblastic.

10.Myelodysplastic Syndrome:

"Preleukemia" as it was formerly called or Myelodysplastic syndrome is fairly an infrequent cause of anemia, but is a more common

cause in the aged individuals than in younger patients. Milman N et al (1994)[37]has shown that about 5.8% of the total anemia population had Myelodysplastic syndrome (MDS) and 17.2 % of the patients had Unexplained anemias. MDS encompass a spectrum of clonal hematopoietic stem cell disorders characterized by cytopenias, ineffective hematopoiesis, cytopenias, clonal chromosomal abnormalities and a risk for acute leukemic transformation in about 30 -35 % of patients. The threshold for cytopenias are hemoglobin less than 10g/dl, Absolute Neutrophil count less than 1.8x 10^9/L and platelets less than 100 x 10^9/L. MDS arising denovo is termed Primary MDS. Primary or denovo MDS predominantly affect older individuals with a median age of onset of 60 – 70 years[38]. Predisposing factors could be genetic or acquired. Genetic factors have a role in Pediatric MDS. Secondary MDS is diagnosed when a known acquired predisposition is documented. This could be due to exposure to benzene, tobacco, radiation or MDS evolving from Aplastic anemia and Paroxysmal Nocturnal Hemoglobinuria (PNH). Therapy related MDS is seen in patients who have received chemotherapy earlier, usually for lymphomas.

MDS have an estimated annual incidence of approximately 3.5 to 10 per 100 000 in the elderly population[39]. The rising frequency and incidence of MDS in the US population may be attributed to the growing aging population and increasing disease recognition and diagnosis. The etiology

and pathogenesis of MDS remain poorly characterized. Underlying the marrow failure and peripheral cytopenias is deranged clonal proliferation of hematopoietic progenitor cells with inherited or acquired genetic mutation. Clonality in MDSs has been supported by cytogenetics, FISH and X chromosome inactivation studies. The early proliferative advantage of MDS clones may be represented by increased cellular proliferation particularly in the myeloid lineage and potentially all hematopoietic lineages. Accumulating multiple alterations that may affect cycle regulation, transcription factors, growth factor receptors and tumor supressors, these clonal cells further expand with abnormal maturation and increased rates of apoptosis. Excessive apoptosis of hematopoietic progenitor cells may contribute to the defining features of MDSs, including dysplastic morphological features, ineffective hematopoiesis and marrow failure. Alongside this observation some studies suggest that disease progression may be accompanied by decreased rates of apoptosis, because susceptibility to intramedullary cell death changes with changing phenotype[40].

Laboratory Diagnosis of MDS:

The diagnosis is established after a careful examination of peripheral blood smear and along with a bone marrow aspirate and/or biopsy. Isolated anemia(Hb < 11g/dl) is the most common presentation in about 85% of cases but rarely, it can present with isolated

thrombocytopenia. An even less common presentation is isolated neutropenia[41] .The anemia can be Normocytic, Macrocytic or Microcytic accompanied by a low reticulocyte count. Other RBC abnormalities that can be observed are anisopoikilocytosis, basophilic stippling, Howell-Jolly bodies, fragmented cells and nucleated red cells. There may be monocytosis, basophilia eosinophilia, or lymphocytosis. Cytoplasmic hypogranularity and hyposegmented neutrophils (pseudo- pelger Huet anomaly) are other characteristic feature. Few circulating Myeloblasts may be seen. Platelets can be large and both hypogranular and hypergranular forms can be seen. *Bone marrow:* Marrow is usually hypercellular. Cellularity may be decreased and in such cases trephine biopsy of marrow is more informative regarding cellularity and percentage of blast cells than a marrow aspirate. The hallmark finding is dysplasia in all three lineages – Dyserythropoiesis, Dysgranulopoiesis and Dysmegakaryopoiesis[42]. Dysplasia should be seen in a minimum of ten percent or greater of the marrow cells of the concerned lineage. The subgroup determination of MDS can be made after taking in to consideration the four features such as the percent bone marrow Blasts, presence of Auer rods, percent of Ringed Sideroblasts, and absolute number of Monocytes in the peripheral blood. Erythroid Lineage: There is megaloblastic erythropoiesis. Features of dyserythropoiesis are distorted nuclear cytoplasmic maturation, nuclear budding, irregular

nuclei, karyorrhexis, intranuclear bridging, multinuclearity. Cytoplasmic abnormalities include vacuolization, basophilia and poor hemoglobinisation. The other characteristic morphologic feature is Ringed Sideroblasts. They have five or more granules/cell and encircling more than one third of nucleus and should be more than 15% in the Refractory Anemia with Ringed Sideroblast subtype of MDS. Erythropoiesis is shifted to left and the number of erythroid precursors varies between five to fifty percent.[43]. *Myeloid series* Myeloid hyperplasia is usually seen in MDS. There is hypogranularity and hyposegmentation of myeloid cells. Other nuclear dysplastic features that can be observed are ring shaped nuclei, nuclear sticks and clumping of chromatin[44] .*Platelet Abnormalities* In the marrow, there is megakaryocytic hyperplasia or the number is normal. The characteristic abnormalities are Micromegakaryocytes, Multinucleated megakaryocytes and Hypolobated megakaryocytes. Hypolobated megakaryocytes are more common in 5q- syndrome. Micromegakaryocytes(dwarf forms), are two times less than the diameter of a neutrophil, and hypolobated megakaryocytes are moderate sized with monolobate eccentric nuclei and hypogranular cytoplasm[45]. *Blast cell characterization in MDS* Identification of Blast cells is most important in classification and prognostication of MDS. The overall survival and overt leukemic progression is determined by the percentage of blast cells in marrow.[46]

Three types of blasts can be recognized that may have prognostic significance. *Type I blasts:* These cells bear a resemblance to pro-myelocytes, have a reticular nuclear chromatin pattern and one to three nucleoli, with a moderately basophilic cytoplasm. Cytoplasmic granules and Auer rods are absent. *Type II blasts:* These cells contain few azurophilic granules and a lower nuclear/ cytoplasmic ratio. *Type III blasts:* these are blast cells with more than or equal to 20 azurophilic granules without a golgi zone.

Table 2: FAB classification of MDS

Morphologic Subtype	***Peripheral Blood***	***Bone Marrow***	***MDS Diagnoses, %***
Refractory anemia (RA)	Blasts <1%	Blasts <5% ringed sideroblasts <5%	10 -40
Refractory anemia with ringed sideroblasts (RARS)	Blasts <1%	Blasts <5% ringed sideroblasts>15%	10 -35
Refractory anemia with excess Blasts(RAEB)	Blasts <5%	Blasts 5-19%	25 -30
Refractory anemia with excess blasts in transformation(RAEB-t)	Blasts > 5% Or Auer rods present	Blasts 20- 29% Or Auer Rods present	10 -30
Chronic myelomonocytic Leukemia(CMML)	Blasts >5% <1 x 109/L monocytes	Blasts <20%	10 – 20

Table 3: WHO CLASSIFICATION OF MDS

MDS Subtype (WHO)	***Peripheral Blood***	***Bone Marrow***	***FAB Subtype***
Refractory cytopenia with unilineage dysplasia(RCUD)	Anemia Absent/rare blasts	unilineage dysplasia <5% blasts <15% ringed sideroblasts	RA
Refractory anemia with ring sideroblasts(RARS)	Anemia Absent blasts	Isolated erythroid dysplasia <5% blasts <15% ringed sideroblasts	RARS
MDS with isolated del 5q	Anemia Blasts <5% Normal to increased platelets	Normal to increased megakaryocytes with hypolobulated nuclei <5% blasts Absent Auer rods Isolated (del)5q	RA
Refractory cytopenia with multilineage dysplasia (RCMD)	Bi/pancytopenia No/rare blasts No Auer rods Monocytes< 1x 10^9/L .	dysplasia in > 10% of the cells of >2 myeloid cell lines <5% blasts No Auer rods <15% ringed sideroblasts	RA
RCMD – RS	Bi/pancytopenia No /rare blasts No Auer rods Monocytes< $1x10^9$/L.	dysplasia in >10% of the cells of >2 myeloid cell lines <5% blasts Absent Auer rods >15% ringed sideroblasts	RARS

Refractory anemia with excess blasts – 1(RAEB -1)	Cytopenias <5% blasts No Auer rods Monocytes <$1x10^9$/L	Unilineage or multilineage dysplasia 5%–9% blasts No Auer rods	RAEB
Refractory anemia with excess blasts – 2(RAEB – 2)	Cytopenias 5%–19% blasts Auer rods Present or absent Monocytes<$1x10^9$/L	Unilineage or multilineage dysplasia 10%–19% blasts Auer rods Present or absent	RAEB
MDS – unclassified	Cytopenias Absent/rare blasts Absent Auer rods	Unilineage dysplasia in granulocytes of megakaryocytes <5% blasts Absent Auer rods	…..

MDS with isolated del 5q syndrome

This is a distinct clinical category within the WHO classification of MDS. This is a disease of elderly women characterized by thrombocytosis with transfusion dependent anemia of macrocytic type which is therapy resistant. Marrow demonstrates large number of hypolobated forms of megakaryocytes with paucity of erythroid precursors. There is mild leucopenia and normal marrow blast count. The clinical course is mild and prognosis is good[47].

Trephine biopsy in MDS[48]

Bone marrow biopsy in MDS is useful to determine cellularity of marrow, Abnormal Localization of Immature Precursors (ALIP), Reticulin fibrosis, Megakaryocytic dysplasia, lymphoid aggregates and

Hypoplastic MDS. There is increase in number of dysplastic early normoblasts and proerythroblasts. The late normoblasts are reduced disturbing the normal maturation. Megaloblasts have regular and uniform nuclei with linear nucleoli and these should not be mistaken for blasts. There is preponderance of early and intermediate megaloblasts and the late megaloblasts demonstrate dyserythropoiesis. Clusters of more than five blasts are abnormally located in the central parts of the marrow (ALIP). Normally the blasts are located in paratrabecular location. The higher the number of ALIPs, the worse is prognosis. Megakaryocytes are increased in number and many are seen in paratrabecular location. Normally they are seen in central parts of the marrow. Micromegakaryocytes are characteristics and CD41/CD61 may be used to confirm. Hypolobation,giant forms, irregular nuclear lobation and maturation are well made out in biopsy.

Marrow demonstrates increased in Reticulin in 20% of the cases and this is associated with increased number of megakaryocytes in the marrow. There is increased apoptosis in MDS. Neovascularisation is significantly increased and Angiogenesis is correlated with transformation to acute leukemia[54].

Differential diagnosis

Dysplastic changes in one or two cell lines are seen in some conditions which may be difficult to differentiate from MDS. The non

malignant hematological conditions that are associated with Myelodysplasia are vitamin B12/folic acid deficiency, exposure to arsenic and other heavy metals, Congenital Dyserythropoietic anemia, Paroxysmal Nocturnal Hemoglobinuria HIV infection parvo virus B19 infection, G-CSF therapy. Hematological malignancies associated with myelodysplasia are Hypoplastic AML, Myeloproliferative disorders, AML – therapy related and AML in the elderly, Idiopathic myelofibrosis, accelerated phase of CML.

Vitamin B12 and folic acid deficiencies display morphologic alterations in all the hemopoietic cell lines and these revert with appropriate therapy. G-CSF therapy induces marked dysplasia of neutrophil series with hyper granularity; Dohle bodies and nuclear hypolobation and blasts may increase up to 10%. HIV infection results in dysplastic hemopoiesis. Lead poisoning leads to microcytic hypochromic anemia with basophilic stippling and Sideroblastic erythropoiesis. HIV associated dysplasia includes dyserythropoiesis, dysmegakaryopoiesis and giant metamyelocytes without hypersegmented neutrophils. Following zidovudine therapy erythropoiesis is megaloblastic and dysplastic changes are greater. However in HIV infection, blasts are not increased.

Variants of MDS[49]

Hypoplastic MDS:

Nearly, ten to fifteen percent of cases of Myelodysplastic syndromes are of hypocellular MDS type with higher prevalence in females. In these cases there are severe cytopenias and cellularity of marrow is <30% in adults who are less than 60 years or less than 20% in those more than 60 years of age. Majority of patients present with Refractory Anemia (RA). The diagnosis of MDS is made when the dysplastic features of megakaryocytes/and/or myeloid cells, excess of blasts and a cytogenetic abnormality is demonstrable.

Hypoplastic MDS needs to be demonstrated from Hypocellular AML and Aplastic anemia. In MDS fibrosis is more and unlike in aplastic anemia megakaryocytes are seen. CD34 is useful for demonstration of blasts. In these cases, response to immunosuppressive therapy is better than in cases with normo/hypercellular marrow. Prognosis is similar to the cases with normo/hypercellular MDS.

Secondary/therapy related MDS:

Alkylating agents, radiation, benzene, toxins and HIV infection cause secondary MDS. The mean age of presentation is 10 years younger than that of primary MDS. Marrow demonstrates normal or increased cellularity and there is striking Trilineage dyspoiesis. There is pronounced pancytopenia and most of the cases are of RAEB type.

Peripheral smear demonstrates marked anisopoikilocytosis and nucleated RBCs.

Trephine biopsy is valuable in differentiating secondary MDS from primary type. Stromal changes like increased reticulin, stromal edema and gelatinous marrow transformation are observed. There may be necrosis of bone and marrow. In secondary MDS other marrow changes include presence of plasma cells, reactive lymphoid nodules and granulomas in the trephine biopsy.

Therapy related MDS are of two types – MDS occurring after many years of alkylating drug intake and are associated with del 7q and/or del 5q abnormalities and MDS resulting after more than two years of use of topoisomerase II inhibitor drugs. Both these subtypes frequently evolve into AML than denovo MDS. Therapy related MDS is very aggressive in its clinical course.

MDS with fibrosis of marrow:

MDS-f is characterized by marked increase in bone marrow reticulin fibres, pancytopenia and minimal organomegaly. They comprise nearly 10% of primary MDS cases. There is trilineage dysplasia with prominent dysmegakaryopoiesis. Majority of the cases demonstrate RAEB type picture. Since there is increased marrow fibrosis, CD34is useful in demonstrating blasts in fibrous tissue; which usually are not discerned in the bone marrow aspirate.

MDS-f should be differentiated from other disease states in which there is marrow dysplasia with fibrosis like AML M7, AML with multilineage dysplasia, primary myelofibrosis and CML in accelerated /blastic phase with marrow fibrosis.

MDS of childhood:

MDS of childhood is relatively rare in comparison to MDS of the elderly. Most of the childhood MDS cases become symptomatic rather early and transform in to AML in a very short span.MDS in children has an aggressive clinical course irrespective of the WHO subtype. There are many differences between MDS in children and adults.e.g. RARS is extremely rare in children. Childhood MDS is more often associated with pre-existing marrow failure or congenital abnormalities like Kostmann's syndrome, Schwachmaann- Diamond syndrome, Diamond- Blackfan anemia, Fanconi anemia, Down's syndrome, Neurofibromatosis I and Juvenile Xanthogranuloma. Juvenile MyeloMonocytic leukemia (JMML) is the commonest. Cytogenetic abnormalities occur in 60-70% of primary MDS in children. In primary MDS, monosomy 7 is the most common cytogenetic abnormality.

Prognosis in MDS:

MDS patients have a reduced life expectancy compared with normal controls. This difference is marked in patients who are less than 60 years. Though the WHO classification of MDS is of prognostic

significance, scoring systems provide objective parameters for improved reproducibility. International prognostic scoring system (IPSS) is computed from three parameters – marrow blast percentage, number of lineage affected and bone marrow cytogenetics. Of the different types of MDS, pure sideroblastic anemia and the 5q- syndrome are associated with an excellent prognosis with a low rate of transformation in to acute leukemia.[50]

Table 4: International Prognostic Scoring System (IPSS)

Risk	score	AML Transformation %	Median survival (years)
Low	0	19	5.7
Intermediate-1	0.5-1.0	30	3.5
Intermediate - 2	1.5-2.0	33	1.2
High	2.5	45	0.4

Management of MDS:

The only treatment is Allogenic stem cell transplantation. Management is individualized and guided by patient's age, prognosis and toxicity of treatment. Low risk MDS is associated with longer survival. Hence amelioration of hematological deficits is the therapeutic goal. Treatment consists of erythropoietin and combined administration of G-CSF and GM-CSF, immunosuppressive therapy, antiangiogenic agents and management of neutropenia. High risk MDS have risk of leukemic

transformation and shorter survival. Hence, prolongation of survival and warring of leukemic evolution is the target. 5- azacytidine, a DNA hypomethylating/ methyl transferase inhibitor is the most promising therapy for improved quality of life in MDS.

11.Unexplained Anemia of Elder People:

A mild, normochromic normocytic anemia with a hemoglobin concentration usually between 11 and 12 g/dL has been observed in people above the age of seventy years. More than thirty percent of the anemias in the older adult might be put under the category of this Unexplained anemia[51]. The pathogenesis of unexplained anemia in geriatric patients is poorly characterized, and it is primarily a diagnosis of exclusion[51]. Several possible mechanisms have been put forth to explain this type of anemia. These include alterations in hematopoietic stem and erythroid progenitor cell number and/or function, hypogonadism, decline in renal function with age and/or a reduction in erythropoietin secretion and finally the presence of early or overt Myelodysplastic syndromes. It is a hypoproliferative anemia and is characterized by a low absolute reticulocyte counts and a low reticulocyte index[52]. Other associated features are low lymphocyte, neutrophil, and platelet counts. One other feature that implies that this condition is not merely a normal age – related variant is the increased red blood cell 2, 3 – DPG level present in these patients[53]. Traditionally, it is thought that in response to hemolytic

anemia, the bone marrow is able to attain a compensatory increase in erythropoiesis of about eight fold in children and about fivefold in adults. However this does not hold true for the older adult. Certain other factors need to be emphasized when taking in to account the unexplained anemias in the elderly. The first is the undetermined potential impact of changes in levels of testosterone or estrogen with age. Next is that, older individuals have a tendency for poly pharmaceutical usage. A variety of these drugs are capable of suppressing erythropoiesis. Lastly, careful inquiring of all considerable co morbid conditions in an older person's medical history is essential in defining and managing unexplained anemia in elderly.

Myelodysplastic syndrome (MDS) is likely to be the cause of anemia in a significant percentage of elder individuals with Unexplained anemia. MDS is primarily a disease of the older adult with a median age at diagnosis of 65 years. Approximately 5-15% of elder patients with unexplained anemia are liable to have Myelodysplastic syndromes using French American British criteria and a further minor subgroup of patients may have abnormalities that are suspicious for, but not confirmatory of MDS[54]. Aditional studies are needed to better categorize these patients, called pre-MDS category and for better understanding their long term prognosis. Emerging molecular techniques to detect clonal hematopoiesis are expected to provide a vital part in this category[55]. Indications for

Bone Marrow Aspiration or Biopsy in these patients are[56] (1) peripheral blood film with immature white cells or nucleated red cells, (2) Pancytopenia, (3) Monoclonal Gammopathy, (4) Suspicion of MDS, (5)Indeterminate status of iron stores or (6)Unexplained progressive or refractory anemia.

12.Myeloproliferative/ myelodysplastic(MPD/MDS) neoplasms

This group of overlap disorders demonstrates features of both myeloproliferation and myelodysplasia and includes chronic myelomonocytic leukemia (CMML), atypical chronic myelogenous leukemia, juvenile myelomonocytic leukemia (JMML) and unclassifiable MPD/MDS neoplasms[57]. *CMML* This clonal neoplasm is characterized by monocytosis persistent for more than three months. There is ineffective hematopoiesis leading to anemia and thrombocytopenia. These cases are bcr/abl negative. Monocytosis is almost always more than ten percent of the white cells. Blasts and promonocytes are less than twenty percent. Bone marrow is hypercellular with myeloid hyperplasia and increased monocytes and promonocytes. Mild degree of dyserythropoiesis and dysmegakaryopoiesis are present. Monocytic series are confirmed with non-specific esterase stain. Reticulin fibrosis is present. The common cytogenetic abnormality is t (5; 12) (q33:p13). *Atypical CML* It is a rare Ph negative MPN. Anemia is more severe than CML. Total count is less than in typical CML and usually in the range of 24-100x10^9 /L.

promyelocytes,myelocytes and metamyelocytes are fewer than in CML and constitute 10-20% 0f WBCs. Monocytosis is more than typical CML but less than in CMML. Platelets are diminished. Bone marrow is hyper cellular with myeloid hyperplasia with multilineage dysplasia. Blasts are less than twenty percent. Patients with aCML have a poor prognosis with median survival of about two years. *JMML* This is a bcr/abl negative distinct entity seen in very young children aged less than two years and is characterized by a prominent monocytic component and is clinically an aggressive disease.

Unclassifiable MDS/MPD neoplasms:

Cases which are in transition and have not evolved into any distinct entity are included in this subgroup. The diagnostic criteria are (i) no preceding history of growth factor or cytotoxic therapy or MPN or of MDS. (ii) myeloproliferative features like platelet count >450x 10^9/L, splenomegaly and TLC > 13x 10^9/L. (iii) hematologic and clinical findings of one of the categories of MDS with <20% blasts in blood/BM.

Leukemias in Elderly

Most Leukemias have a predilection for the adult age group possibly with the exception of Acute Lymphoblastic Leukemia (ALL). In elderly, the commonest types of leukemia are Acute myeloid Leukemia (AML), Chronic Lymphocytic Leukemia, Chronic Myeloid Leukemia and Acute Lymbhoblastic Leukemia. Other less common forms are Hairy

cell leukemia, Large Granular Lymphocytic Leukemia and Adult T-cell Leukemia. *AML:* The incidence of Acute myeloid leukemia increases with age. The median age group is around sixty seven years. Men are more commonly affected. The subtypes of AML common in elderly age group are erythroleukemia (FAB M6), acute megakaryoblastic leukemia (FAB M7) and the rare acute panmyelosis with myelofinrosis.anemia and thrombocytopenia are present in nearly all cases[58]. In aleukemic leukemia total count is low. In bone marrow, blasts constitute more than 20% of non erythroid cells of marrow. Auer rods are the most reliable morphological feature of AML. These are prominent in AML M2 and AML M3. *AML-M6*: this is rare leukemia constituting only 3-4% of all cases of AML. The criteria for AML- M6 are (i) erythroblasts more than 50% of bone marrow nucleated cells (ii) blasts more than 20% of bone marrow non erythroid cells. In pure erythroid leukemia the early erythroblasts are more than 80% of the marrow cells. AML-M7: it is an uncommon form of AML accounting for 8-10% of all cases in adults and 2-3% of cases in children. It is not an uncommon form of blast crisis in CML since it occurs in nearly 20% of Ph positive CML cases. AML has a dismal prognosis in the elderly. This could be due to the following reasons.

(i) Elder patients are unable to tolerate the intensive cytotoxic chemotherapy.

(ii) They have a higher incidence of co morbid disorders, poor tolerance to infections and a poor organ reserve.

(iii) It frequently arises from antecedent MPD or MDS which is common in elderly and associated with poor prognosis.

(iv) Higher incidence of unfavourable risk cytogenetics in elderly such as inv(3), t(11:19), t(6:9), del5, del 7.[59]

ALL: Although commoner in children, a second peak is usually seen after the age of fifty years for ALL. The incidence is 1.0 to 1.6/ 100,000. Advanced age along with increased leucocyte count, immunophenotype and cytogenetics is a poor prognostic factor for ALL. The Philadelphia positivity status rises with increasing age in these patients which is associated with a poor prognosis[60]. *CLL:* It usually affects adults over the age of fifty five years. It is a low grade malignancy with a survival rate of seventy five percent. *CML:* This is the most common leukemia encountered in India. It is a clonal myeloproliferative neoplasm arising from neoplastic transformation of stem cell resulting in overproduction of normal appearing granulocytes. The hallmark finding is the Philadelphia chromosome t (9: 22). CML passes through a triphasic course of (i) chronic phase of massive leucocytosis (ii) accelerated phase with sudden rise in blast cells and clinical deterioration and (iii) blast

crisis with blasts more than twenty percent in bone marrow with rapid deterioration clinically. Its incidence increases with age with more than 50% patients diagnosed at more than or equal to sixty years of age[61]. Survival rate is above ninety percent. Peripheral smear: there is marked leucocytosis with total count ranging from 30x 10^9/L to 500 x 10^9/L.neutrophils and myelocytes are the predominant cells in chronic phase. Basophils are more in accelerated phase. Presence of tear drop cells and anisopoikilocytosis suggests fibrosis of the marrow. Thrombocytosis varies from 300 – 600 x 10^9/L. neutrophil alkaline phosphatase score is markedly diminished to 0 – 20. Bonemarrow: marrow is hypercellular with replacement of fat by hyperplastic hemopoietic cells with marked myeloid hyperplasia and an elevated M: E ratio. Blasts are more than 20% in blastic crisis. Pseudo- Gauchers cells are macrophages with granular cytoplasm scattered among the marrow cells formed due to increased lipid turnover from granulocytic membranes and accumulating in the macrophages. Differential diagnosis: (i) leukemoid reaction: in CML, marrow is hypercellular with loss of fat, while fat spaces are made out in the marrow in a case of leukemoid reaction. Lack of basophilia and eosinophilia with high NAP score is suggestive of a leukemoid reaction. (ii) Essential thrombocythemia – there is marked megakaryocytic hyperplasia with myeloid and erythroid proliferation. Clinical picture, blood picture and bcr/abl fusion gene are

taken in to account to differentiate essential thrombocythemia from CML.(iii) cellular phase of idiopathic myelofibrosis- marrow is hypercellular with hyperplasia of all the three cell lines with normal maturation. Megakaryocytic proliferation is more marked with many immature and dysplastic forms present in clusters.

MATERIALS AND METHODS

This prospective study was conducted in the Department of Pathology, Tirunelveli Medical College & Hospital under due approvals of the TVMC Institutional Research Ethics Committee. Patients were sourced from the Departments of General Medicine, General Surgery, Oncology and Geriatric Medicine over a period of two years from September 2010.

Geriatric patients of age more than 60 years who are presenting with anemia, fulfilling the WHO criteria of anemia (Hemoglobin <13gms/dl in males, <12gms/dl in females) were enrolled in the study. Sixty five patients were selected non consecutively. Consent was taken from all patients. The following were taken as the Inclusion Criteria: namely (1) Patients more than 60 years of age (2) Patients of all gender predispositions (3) Patients with anemia as per WHO standards. The patients were excluded as per the following Exclusion Criteria: (1) Patients with a history of recent transfusion (2) Patients who have undergone major surgical procedure in the past 3months. (3) Acute and Terminally ill patients.

Sixty five patients who satisfied our selection criteria were selected for the study. A detailed clinical interview was undertaken and all details regarding age, gender, occupation, racial characters, socio economic milieu, presenting complaints, previous history as pertaining to

the presenting complaints, history of exposure to chemicals or drugs, dietary habits, co-morbid conditions, were recorded. A detailed clinical examination was done and presence of organomegaly, lymphadenopathy, clubbing, cyanosis, pedal edema etc as per protocol was recorded.

A standardized set of lab investigations were ordered for all these patients - Complete Blood Count, Basic Clinical Chemistry, Liver Function Tests, Thyroid Profile, Reticulocyte Count and Peripheral Blood Film. Radiological investigations such as roentgenogram, sonographic studies, CT scans and MRI were performed based on clinical needs. Some patients were given an endoscopic analysis of the upper and lower GIT as clinically required.

All the patients who were selected for the study were advised a bone marrow aspiration study as per standard clinical protocols of our hospital. It was advised in patients with blood smear showing microcytic, normocytic, macrocytic or dimorphic blood picture, cytopenia, and abnormal cellular morphology. Bone marrow was aspirated from the sternum or the posterior superior iliac crest, using a Salah needle. Aspirated were used to make 8-15 smears. The smears were air-dried, fixed in methanol and stained with Leishman's stain as per standard protocol. One smear was saved for perl's stain. The Bone marrow samples were examined and the findings were recorded in a standard format. The smears were assessed for (1)Degree of Cellularity - graded

as hypocellular, normocellular or hypercellular (2) Relative distribution of erythroid, myeloid, and lymphoid cells (3) Morphological abnormalities of the erythroid, granulocytic, and megakaryocytic series (4) Presence of other cells. The finding was recorded.

The results were tabulated and statistically analyzed.

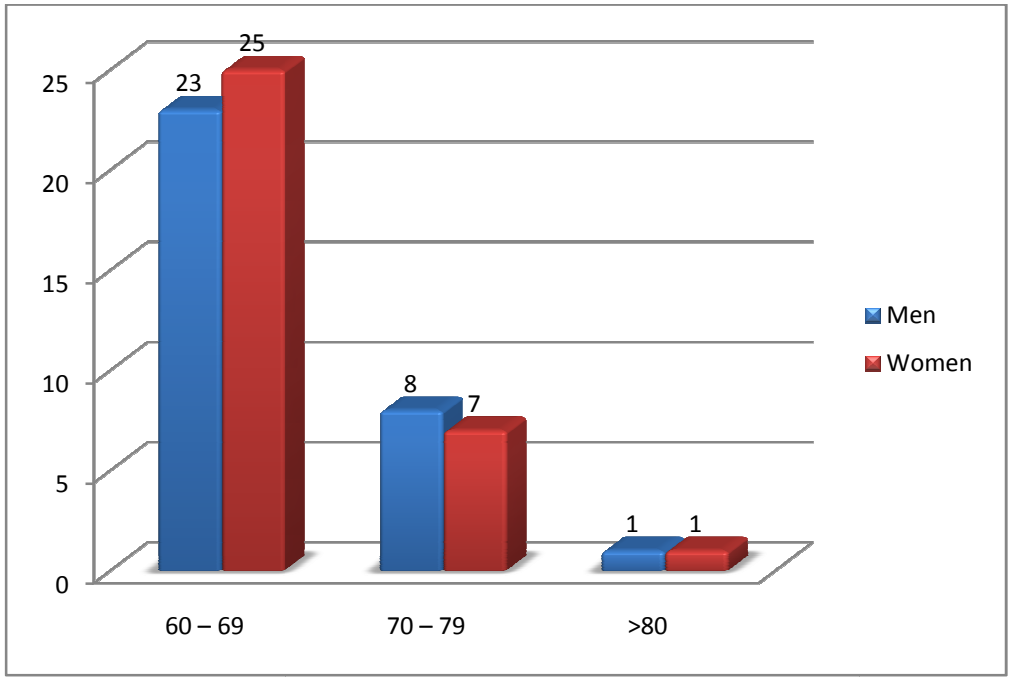
OBSERVATIONS AND RESULTS

This present study was a prospective study carried out in the Department of Pathology, Tirunelveli Medical College for a period of two years. A total of sixty five patients above 60 years of age with anemia were evaluated, the results analyzed and tabulated.

Table 5 Age and Gender Distribution of Patients

Age in years	No Of Patients			Percentage
	Men	Women	Total	
60 – 69	23	25	48	73.84
70 – 79	8	7	15	23.08
>80	1	1	2	3.08
Total	31	34	65	100

Chart 1 Age and Gender Distribution of Patients



The age of the patients enrolled in our study was between 60 to 85 years. The mean age was 66.65 years. Most of the patients were in the age group of 60–69 years (73.84%). 23.08% patients were between 70-79years and 3.08% above 80years of age. (Table 5) .Of the 65 patients, 34 (52.31%) were women and 31 (47.69%) were Men. 22 men and 26 women were between 60 – 69 years, 8 men and 7 women were between 70-79 years.(Table 5)(Chart 1)

Table 6 Distribution of the Presenting Complaints

Symptoms	No of Patients	Percentage
Fatigue	24	36.92
Dyspnoea	19	29.23
Pedal edema	13	20.00
Abdominal pain	9	13.85
Neurological	3	4.62
GI Bleed	3	4.62
Others	8	12.31

The most common presenting complaint was Fatigue found in 24(36.92%) patients. The next common symptom was Exertional dyspnoea which was present in 19(29.23%) patients. These were followed by pedal edema and abdominal pain which was present in 13(20%) and 9(13.85%) patients respectively. 3(4.62%) patients presented with neurological symptoms and another 3(4.62%) patients with bleeding diathesis. Other less common symptoms accounted for

12.31%. Many patients had overlapping symptoms amongst these. (Table 6)

Table 7 Co-morbid Conditions in the Study Group

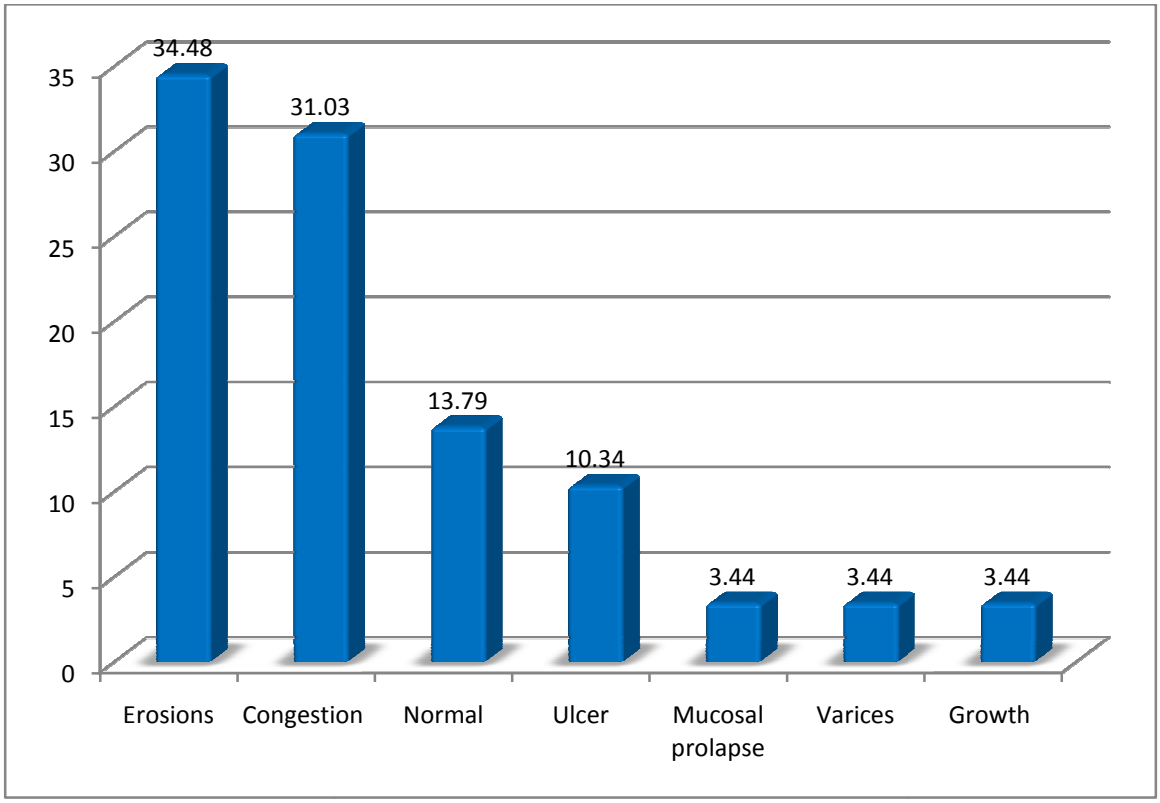
Condition	No Of Patients	Percentage
Hypertension	21	32.31
Diabetes	12	18.46
Renal disease	7	10.77
Tuberculosis	6	9.23
Bleeding diathesis	6	9.23
CAHD	5	7.69
Malignancy	5	7.69
Liver disease	4	6.15
APD	2	3.08
Hypothyroid	1	1.54

The co-morbid conditions associated with our study group were Hypertension(HT) seen in 21(32.31%) patients, Diabetes mellitus(DM) in 12(18.46%) patients and Renal disease in 7(7.69%) patients. 6(9.23%) patients had a history of Tuberculosis(PT) and another 6(9.23%) patients had a history of gastrointestinal and/or genitourinary bleed. An H/O coronary artery heart disease (CAHD) was present in 5(7.69%) patients. Liver disease was present in 4(6.15%) patients. H/O of solid organ malignancy was present in 5(7.69%) patients, 2(3.08%) had H/O Acid Peptic Disease (APD) and 1(1.54%) patient was a hypothyroid. (Table 7). Overlapping features were present in many patients.

Table 8 UGI Endoscopy Findings

Endoscopic Findings	No of patients	Percentage
Mucosal Erosions	10	34.48
Congestion	9	31.03
Normal	4	13.79
Ulceration –Stomach	3	10.34
Mucosal Prolapse	1	3.44
Grade II Varices	1	3.44
Growth	1	3.44
Total	29	100

Chart 2 UGI Endoscopy Findings

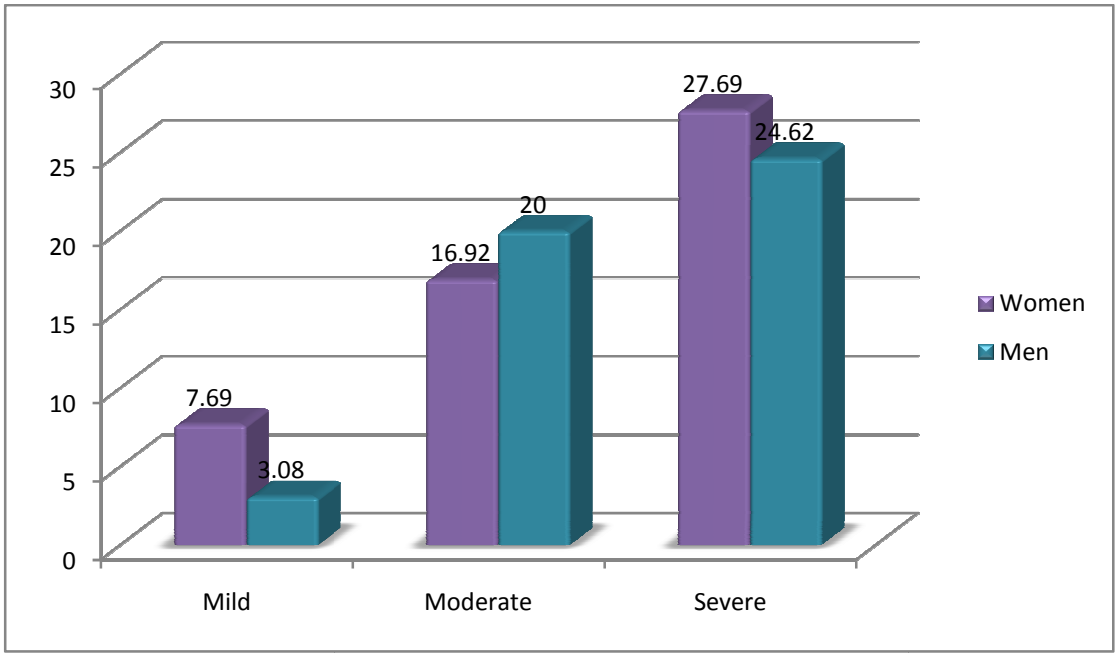


Out of the total 65 patients, 29 patients underwent Upper Gastro intestinal endoscopy. Endoscopy revealed normal findings in 4(13.79%) patients, mucosal erosions in 10(34.48%) patients, congestion in 9(31.03%patients) and gastric ulcers in 3(10.34%) patients. Esophageal varices was present in 1(3.44%) case, an ulceroproliferative gastric growth in 1(3.44%) patient and mucosal prolapse in 1(3.44%) of our patients. (Table 8)(Chart 2).

Table 9 Severity of Anemia

Severity	**Gender**	**No of Patients**	**Total**	**Percentage**
Mild	Men	2 (3.08%)	7	10.77
	Women	5 (7.69%)		
Moderate	Men	13 (20%)	24	36.92
	Women	11 (16.92%)		
Severe	Men	16 (24.62)	34	52.31
	Women	18 (27.69)		
Total			65	100

Chart 3 Severity of Anemia

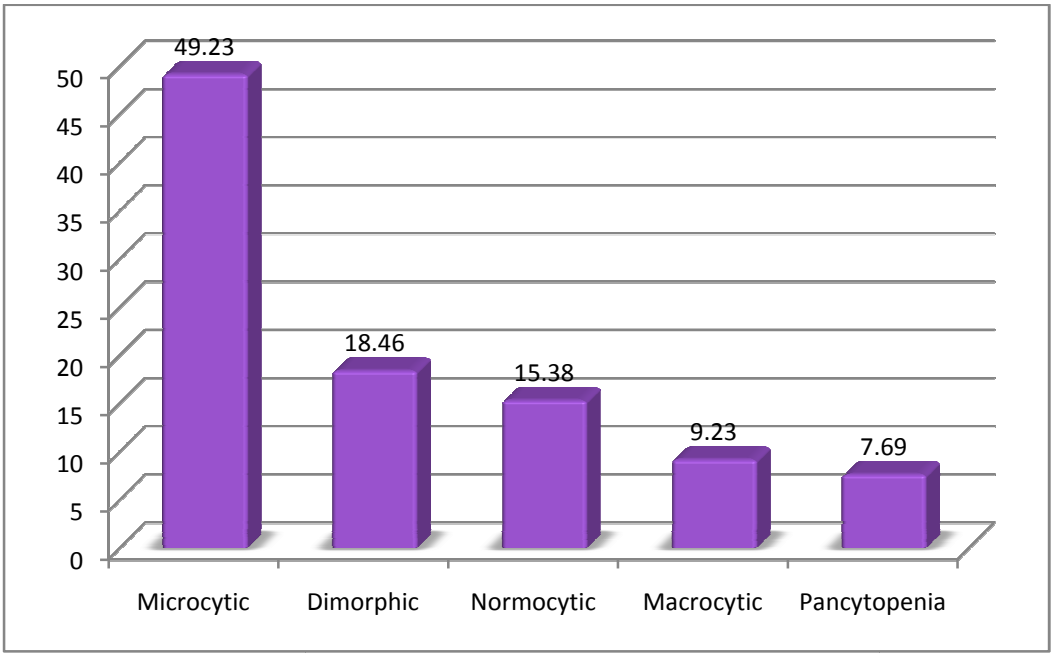


Hemoglobin estimation done in all patients revealed mild anemia in 7(10.77%) patients, a moderate degree of anemia in 24(36.92%) patients and severe anemia in 34(52.31%) patients. Out of the 7 cases of mild anemia, 5 were women and 2 were men. Among the 24 cases of moderate anemia, 13 were men and 11 were women. Out of the 34 cases of severe anemia, 16 were men and 18 were women. (Table 9)(Chart 3).

Table 10 Peripheral Smear Findings

Type of anemia	No of patients	Percentage
Microcytic	32	49.23
Dimorphic	12	18.46
Normocytic	10	15.38
Macrocytic	6	9.23
Pancytopenia	5	7.69
Total	65	100

Chart 4 Peripheral Smear Findings



Peripheral smear examination was carried out in all the sixty five patients. Microcytic hypochromic anemia (MCHC) was observed in 32(49.23%) patients, Dimorphic anemia (DA) in 12(18.46%) patients, a Normocytic blood picture (NA) in 10(15.38%) patients and a Macrocytic blood picture in 6(9.23%) patients. Pancytopenia was observed in 5(7.69%) patients. (Table 10)(Chart 4).

Table 11 Bone Marrow Cellularity

Marrow Cellularity	No of Patients	Percentage
Normocellular	30	46.15
Hypocellular	7	10.77
Hypercellular	28	43.08
Total	65	100

Bone marrow aspiration was done in all patients. It showed Normocellular marrow in 30(46.15%) patients, Hypocellular marrow in 7(10.77%) patients and Hypercellular marrow in 28(43.08%) patients. (Table 11).

Table 12 BM Diagnosis

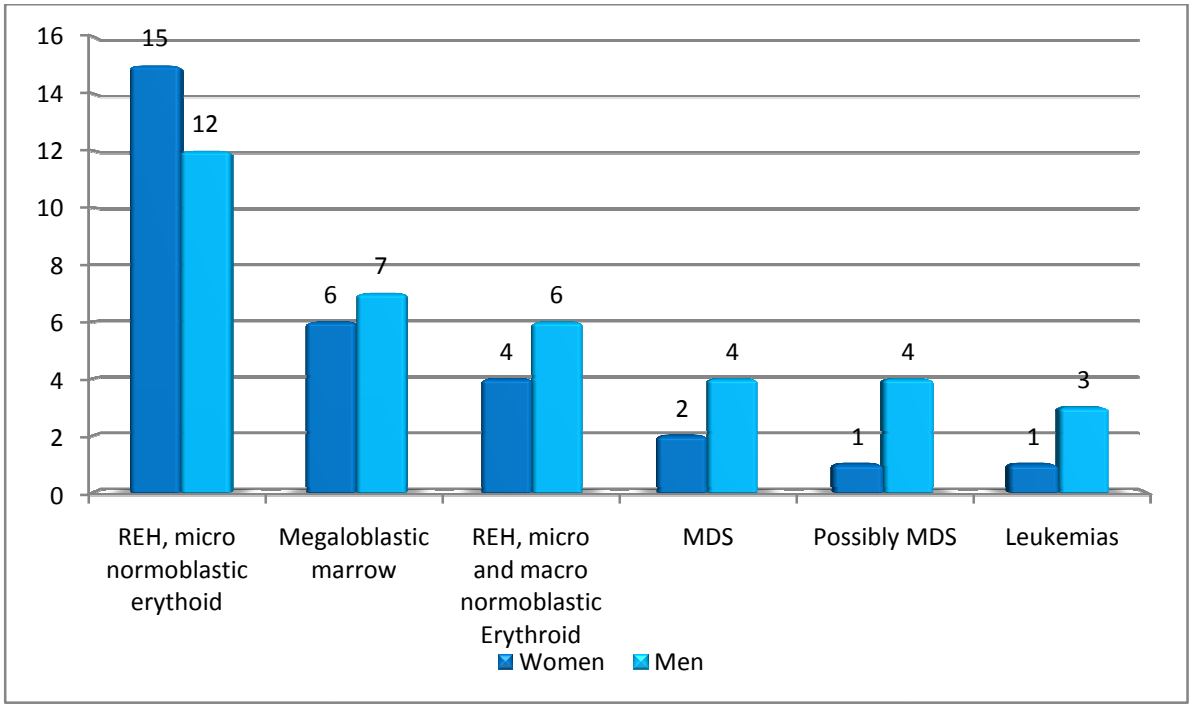
Diagnosis	No of cases	Percentage
Non-malignant Hematological Disorders	55	84.62
Hematologic malignancies	10	15.38
Total	65	100

Overall, non- malignant hematologic disorders were observed in 55(84.62%) patients and hematologic malignancies were observed in 10 (15.38%) patients. (Table 12)

Table 13 Spectrum of Bone Marrow Aspiration Findings

S.No	Marrow Findings	Men	Women	Total	%
1.	Reactive Erythroid wth Hyperplasia of Marrow Micronormoblastic Erythroid Maturation	12	15	27	41.54
2.	Megaloblastic Marrow	7	6	13	20.0
3.	Reactive Erythroid with Hyperplasia of Marrow Micro and Macronormoblastic Erythroid Maturation	6	4	10	15.38
4.	Megaloblastoid Marrow with Unilineage dysplasia. (MDS)	1	0	1	1.54
5.	Megaloblastoid Marrow with. Bi/multilineage dysplasia	3	1	4	6.15
6.	Megaloblastoid Marrow with Multilineage Dysplasia and Excess of Myeloblasts	0	1	1	1.54
7.	Acute Meylogenous Leukemia AML M1	0	1	1	1.54
8.	Chronic Myelogenous Leukemia (Chronic Phase)	0	1	1	1.54
9.	Chronic Myelogenous Leukemia (Accelerated Phase)	1	1	2	3.08
10	Possibly MDS	1	4	5	7.69
	Total	31	34	65	100

Chart 5 Spectrum of Bone Marrow Aspiration Findings



Bone marrow examination showed Reactive Erythroid Hyperplasia (REH) of marrow with micronormoblastic maturation in 27(41.54%) patients. Among them, 12 were men and 15 were women. Megaloblastic marrow changes were observed in 13(20%) patients. Among them, 7 were men and 6 were women. Reactive Erythroid with Hyperplasia of marrow micro and macronormoblastic erythroid maturation was observed in 10(15.38%) patients. Among them 6 were men and 4 were women. Megaloblastoid marrow with unilineage dysplasia was seen in 1(1.54%) male patient, Megaloblastoid marrow with bi/ multilineage dysplasia in 4(6.15%) patients. Among them 3 were men and 1 was a woman. Megaloblastoid marrow with multilineage dysplasia and excess of blasts was seen in 1(1.54%) female patient. Acute Myeloid leukemia was

diagnosed in 1(1.54%) female patient, Chronic Myeloid leukemia in 3(4.63%) patients. Among them 1 was a male and 2 were women. A possibility of a probable MDS was suggested in 5(7.69%) patients. (Table 13)(Chart 5).

Table 14 Comparison of Co morbidities and bone marrow diagnosis

Co-morbid Conditions	REH micronormo blastic maturation	REH Micro and macro Normo blastic maturation	Megaloblastic anemia	MDS	Leukmia	P/O MDS
HT	9	3	5	3	-	1
DM	4	1	3	2	1	1
CAHD	2	1	--	1	-	1
Renal Disease	3	--	3	-	1	-
Liver Disease	2	1	1	-	-	-
Old PT	3	1	2	..	-	-
APD	…	…	1	1	-	-
Hypothyroid	1	-	-	-	-	-
Malignancy	3	--	1	-	-	1

In patients with Reactive Erythroid Hyperplasia (REH) of marrow with micronormoblastic maturation, Hypertension was present in 9 patients. Diabetes mellitus was present in 4 patients. A H/O GI/GU bleed was present in 5 patients of which one had oesophageal varices on endoscopy. H/O Renal disease was present in 3 patients and a H/O Liver disease was present in 2 patients. Of these two, 1 had evidence of hemolysis. UGI endoscopy detected malignancy in 1 patient. H/O pulmonary tuberculosis (PT) was present in 2 patients. Associated solid

organ malignancies were present in 3 cases and they had H/O chemotherapy/ radiotherapy. (Table 14)

In patients with reactive erythroid hyperplasia (REH) of marrow, with micro and macro normoblastic maturation. Hypertension was present in 3 patients. H/O Diabetes and CAHD was present each in one patient. A H/o liver disease was present in one patient. (Table 14)

In patients with megaloblastic anemia, 5patients had h/o hypertension, 3 had Diabetes mellitus and one had a h/o CAHD. One patient was hypothyroid. One patient had H/O chemotherapy for carcinoma breast. Renal parameters were elevated in three patients and liver disease was present in one patient. 2 patients had h/o old PT, and I patient had APD. (Table 14)

Out of the 6 patients with the diagnosis of MDS, no underlying liver or renal disease were found. The other associated co morbid conditions in these patients were CAHD in 1 patient, DM in 2 and HT in 3 patients. (Table 14). In the patients with acute and chronic leukemia, an associated renal disease was present in one patient and DM in one patient. Among 5 patients with the diagnosis of a possibility of MDS, one patient was a case of carcinoma breast with H/O chemotherapy. One had a history of diabetes and treatment for PT and another one patient was a known case of CAHD. (Table 14)

Table 15 Comparison of the RBC Morphology and MCV in patients with Iron Deficiency Anemia

Sl. No	Anemia Grade	Hb g%	MCV	Retic %	WBC x10^3/mm^3	Platelet Count x10^5/mm^3	Peripheral Smear	Iron Stores
1	I	10.3	88.7	1.5	6.5	2.98	Normocytic	Absent
2	III	6.5	72.8	1	4.2	1.1	Normocytic	Absent
3	I	10.4	97.4	1	5.8	90	Normocytic	Absent
4	I	11.2	77.6	0.5	3.8	1.06	Normocytic	Absent
5	II	7.7	86.6	0.5	4.1	1.52	Normocytic	Absent
6	III	6.3	93.6	3	4.2	0.41	Dimorphic	Absent
7	II	7.5	86.4	1.3	7.3	1.50	MCHC	Absent
8	I	10.2	66.6	0.5	6.6	3.32	MCHC	Absent
9	II	8.9	78.9	1.2	6.3	4.32	MCHC	Absent
10	III	6.2	68.7	0.5	5.2	2.13	MCHC	Absent
11	II	8.3	56.5	1	7.3	2.65	MCHC	Absent
12	III	6.4	72.5	1	11	0.19	MCHC	Absent
13	III	5.7	73.5	0.6	6	3.8	MCHC	Absent
14	III	5.8	66.4	<0.5	10.6	3.55	MCHC	Absent
15	II	8.7	72.9	1.1	9.9	4.1	MCHC	Absent
16	III	5.7	76.9	0.5	9.2	1.48	MCHC	Absent
17	II	7.4	77.2	1	4.7	3.96	MCHC	Absent
18	II	7.1	65.5	0.5	5.4	2.31	MCHC	Absent
19	III	6.3	92.7	0.5	4.3	1.35	MCHC	Absent
20	III	5.3	64.3	0.4	5	2.98	MCHC	Absent
21	III	5.9	63.2	1.3	7.8	2.95	MCHC	Absent
22	III	6.8	79	1	6.4	3.35	MCHC	Absent
23	III	6.4	73.4	0.5	5.3	2.05	MCHC	Absent
24	II	8.2	69.6	0.6	5.6	2.29	MCHC	Absent
25	III	6.9	74.8	1	6.1	3.41	MCHC	Absent
26	III	5.7	62.9	0.5	2.9	1.13	MCHC	Absent
27	III	6.3	59.7	0.3	5.3	2.73	MCHC	Absent

The 27 patients with REH with micronormoblastic maturation were given a diagnosis suggestive of a probable Iron deficiency Anemia. (IDA). There was a severe anemia in 15 cases, moderate degree of anemia in 8 cases and a mild degree of anemia in 4 cases. MCV was normal in 6 cases, < 80fl in 21 cases and increased in none. Reticulocyte count was normal in 25 cases, decreased in 2 cases. Peripheral smear showed microcytic hypochromic blood picture in 21 cases, normocytic in 5 cases and dimorphic anemia in 1 case. The marrow was hypercellular in 8 cases and normocellular in 19 cases. Perl's stain showed absent iron stores in all cases. (Table 15)

Table 16 Comparison of the RBC Morphology and MCV in patients with Combined Deficiency Anemia

No	Anemia Grade	HB g%	MCV	Retic %	WBC $X10^3/mm^3$	Platelet Count $x10^5/mm^3$	Blood Film	Iron Stores
1	III	5.6	89.3	0.3	6.9	2.5	Dimorphic	Low
2	III	6.2	74	1	10.5	1.8	MCHC	Low
3	III	5.1	70.8	2	7.3	1.42	MCHC	Low
4	II	9.3	87.4	0.5	4.7	3.16	Normocytic	Low
5	I	10.8	90.4	0.5	5.3	2.84	Normocytic	Normal
6	II	7.5	83.2	1.4	7.8	2.78	MCHC	Normal
7	II	8.3	86.5	0.5	6.5	3.26	MCHC	Low
8	II	8.7	82.6	1.6	7.4	2.53	Dimorphic	Low
9	I	10.4	88.3	1	4.9	1.79	MCHC	normal
10	II	6.8	81.7	1.2	6.3	1.85	Dimorphic	Low

The 10 patients with REH with micro and macro normoblastic maturation were given a diagnosis as suggestive of a probable combined B12/ iron deficiency. The anemia was severe in 3 patients, moderate in 5 cases and mild in 2 cases. MCV < 80fl in 2 patients was observed in cases and was normal in 8 cases. Reticulocyte count was normal in 7 cases and decreased in 1 case and increased in 2 cases. Blood film showed microcytic hypochromic picture in 5 cases, dimorphic picture in 3 cases and normocytic in 2 cases. Marrow was normocellular in 6 cases, hypercellular in 4 cases. Perl's stain showed low iron stores in 7 cases, normal iron stores in 3cases (Table 16)

Table 17 Comparison of the RBC Morphology and MCV in patients with Megaloblastic Anemia

No	Anemia Grade	HB g%	MCV	Retic %	Platelet Count	WBC $x10^3/mm^3$	Blood Film	Iron Stores
1	II	8.1	116.9	1.5	1.73	11.4	Macrocytic	increased
2	II	7.6	84.7	0.5	2.45	4.8	Normocytic	Increased
3	II	8.3	98.4	0.5	2.11	6.4	Dimorphic	Low
4	III	6.1	102.4	0.5	1.5	6.2	Macrocytic	Normal
5	II	9.1	89.4	<0.5	3.3	12	Dimorphic	Normal
6	III	6.6	94	1	2.69	4.3	Macrocytic	increased
7	II	8.2	99.6	0.5	2.31	4.5	Dimorphic	Increased
8	III	4.8	83.3	0.5	0.71	2.9	Dimorphic	Normal
9	II	9.1	100.4	1	2.94	5.9	Dimorphic	Increased
10	III	6.8	92.5	0.5	0.9	3.2	Pancytopenia	Increased
11	II	7.0	66	<0.5	4.22	4.5	Macrocytic	Increased
12	II	7.7	104	0.5	1.27	1.2	macrocytic	Normal
13	III	6.8	128	1.2	0.4	3.2	pancytopenia	Increased

Of the 13 patients with megaloblastic anemia, the anemia was severe in 5 cases and of moderate degree in 8 cases. MCV was >100 fl in 5 cases and was normal in 7 cases and decreased in 1 case. Reticulocyte count was normal in 11 cases, decreased in 2 cases. Blood film revealed dimorphic picture in 5 cases, macrocytic in 5 cases, normocytic in 1 case pancytopenia in two cases. The marrow was hypercellular in 8 cases and normocellular in 5 cases. Perl's stain showed increased iron stores in8 cases and normal iron stores in 4 cases. (Table 17)

Table 18 Comparison of the RBC Morphology and MCV in patients with MDS

No	Grade	HB g%	MCV	Retic	WBC count $x10^3/mm^3$	Platelet count $x10^5/mm^3$	Blood Film	Iron Stores
1	III	4.9	129.3	1	2.5	0.3	Pancytopenia	Increased
2	III	4.7	90.1	0.5	4	0.105	Pancytopenia	Normal
3	III	6.5	91	1	3.2	0.33	Pancytopenia	Increased
4	III	5.4	132.4	1.5	11.9	3.54	MCHC	Increased
5	III	6.8	65.3	0.5	5.8	2.92	Dimorphic	Increased
6	III	5.6	76.4	1	3.9	2.12	Dimorphic	Increased

Out of the 6 patients with MDS, the anemia was severe in all patients. MCV normal in 2 patients, <80fl in 2 patients and >80fl in 2 patients. Reticulocyte count was normal in all cases. Peripheral smear showed a pancytopenic blood picture in 3 cases, a dimorphic picture in 2 cases and a microcytic hypochromic anemia in 1 patient. The marrow was

hypercellular in 4 patients and hypocellular in 2 patients. Perl's stain showed increased iron stores in 5 patients and normal iron stores in one patient. (Table 18)

Table 19 Comparison of the RBC Morphology and MCV in patients with Anemia: Possibly due to AML

No	Severity Grade	HB Value	MCV	WBC Count $x10^3$cells/mm^3	Platelet Count $x10^5$cells/mm^3	Blood Film
1	III	6.9	85.9	145	0.28	MCHC Anemia/

The one patient with possible AML was a female with severe anemia. MCV was decreased. Blood film was suggestive of acute leukemia with Microcytic Hypochromic anemia and Thrombocytopenia. (Table 19)

Table 20: Comparison of the RBC Morphology and MCV in patients with CML

No	Severity Grade	HB g%	MCV	WBC Count $x10^3$cells/mm^3	Platelet Count $x10^5$cells/mm3	Blood Film
1	II	7.7	83.3	125	3.19	MCHC Anemia
2	III	6.7	106	153	3.24	Macrocytic Anemia
3	II	8.9	63.7	90	1.74	MCHC Anemia

Of the 3 patients with CML, the anemia was of moderate degree in 2 cases and was severe in one case. MCV was normal in one, increased in one and was reduced in the remaining one patient. Peripheral smear showed Microcytic hypochromic anemia in 2 cases and was Macrocytic anemia in one patient. One patient was in chronic phase and 2 were in accelerated phase. (Table 20)

Table 21: Comparison of the RBC Morphology and MCV in patients with Anemia: Possibly due to MDS

No	Grade	HB g%	MCV	Retic %	WBC Count $x10^3$cells/ mm^3	Platelet Count $x10^5$cells/mm3	Blood Film
1	III	5.9	78.2	2	7.8	2.3	Dimorphic
2	II	9.1	107.3	0.5	3.7	1.93	Dimorphic
3	I	11.1	97.5	1	5.4	3.15	Normocytic
4	III	6.6	72.3	0.5	8.6	2.64	Microcytic
5	II	7.9	86.6	0.5	9.1	2.32	Microcytic

Of the five patients with the possible diagnosis of MDS, the anemia was severe in two patients, moderate in two patients and was of mild degree in one patient. MCV was decreased in two patients, normal in two patients and increased in one. Reticulocyte count was increased in one and normal in the remaining four patients. Total count was normal in three and increased in two patients. Platelet count was normal in all five. Peripheral smear showed dimorphic anemia in two cases, microcytic in

two and normocytic anemia in one patient. Bone marrow was hypocellular in three patients and hypercellular in two patients.

DISCUSSION

Anemia is common in older age group. A cause for anemia needs to be determined in each patient. The etiology of anemia in elderly is often multifactorial and is due to Nutritional deficiency in one-third of cases, Anemia of Chronic disease in one-third and Unexplained anemia in remaining one-third. In elderly, a Bone marrow examination is required for the diagnosis and management of Hematological disorders. In the present study we analyzed the Bone marrow findings and the clinical and laboratory parameters of geriatric patients with anemia.

A total of 65 geriatric patients with anemia were evaluated in our study. The patients were in the age group between 60 to 85 years with a mean age of 66.65 years.(Table 5). The majority of the patients were between 60-69 years. This is similar to the observations of Bhasin A et al.,(2011)[62] and Choi CW et al.,(2004)[63] where the mean age was found to be 70.51 years and 70 years respectively.

In the present study, Anemia was observed in 52.31% of women.(Table 5). This is in concordance with Petrosvan I et al.,(2012)[64] who showed 52.6% prevalence in women. The present study recorded 47.69% men with anemia. Ania BJ et al.,(1997)[65] and Beghe C et al.,loc.cit.(2004) showed a similar observation whereas Bhasin A et al.,loc.cit.(2011) in their study showed a higher percentage (52%) of men with anemia.

The most common presenting complaint in the present study was fatigue (36.92%), followed by exertional dyspnoea (29.23%) (Table 6). The presenting signs and symptoms in our study were usually attributed to anemia with or without congestive heart failure.

In the present study, the anemia was severe in majority (52.31%) of the patients.(Table 9). Artz AS et al.,(2011)[66] and Elzen.et.al.,(2008)[67] had shown a lower percentage of patients with severe anemia. Mild anemia was observed in only 10.77% of patients in the present study. In contrast, Mauro T et al.,(2010)[68] and Elzen WP et al.,loc.cit(2008) had shown a higher (27.4%) percentage of mild anemias in elderly.

Anemia is a disease of multiple causation and its scientific management requires an exact classification which can be done only after a complete study of blood. A complete blood count including the reticulocyte count and a peripheral blood film examination is an initial indispensable tool in the evaluation of anemia. They have a decisive role in the diagnosis and classification of anaemia.

In the present study, Normocytic anemia was found in 15.38% of patients.(Table 10). This contrasts with those of Elis et al.,loc.cit(1996) and Ania et al.,loc.cit(1997) who had shown a higher percentage of Normocytic anemia was in elderly indiviuals . The present study showed Microcytic Hypochromic anemia as the most common type of anemia found in 49.23% of patients. Bhasin A et al.,loc.cit (2011) and

Mauro T et al.,loc.cit (2010) had shown that Microcytic anemia accounted for 30% and 16.9% of the anemic individuals respectively in their studies. A higher occurrence of Microcytic Hypochromic anemia in our study could be attributed to the increased number of patients from rural areas and of low socio-economic status.

Bone marrow studies, though invasive is a safe and simple procedure and is the most frequently carried out investigation in the evaluation of hematological disorders. In geriatric patients Bone marrow examination is important particularly in the diagnosis of Myelodysplastic syndromes.

The cellularity of marrow decreases with age. In the present study, Bone marrow examination of all the 65 geriatric patients with anemia showed normocellular marrow in 46.15%, hypercellular marrow in 43.08% and hypocellular marrow in 10.77%.(Table 11).

In the present study, the Bone marrow aspiration findings were broadly categorized into malignant and non- malignant hematological disorders. Non-malignant hematological disorders formed the major group comprising 84.62%.(Table 12). The majority of cases was of REH of Marrow Micronormoblastic Erythroid Maturation, suggestive of a probable IDA and was present in 41.54% of patients. In the study by Bhasin A et al.,loc.cit(2011) IDA accounted for 69% of patients which is higher when compared to the present study.

In our study, in patients with REH of marrow, Normocytic anemia was seen in 5(18.52%) patients.(Table 15). Apart from iron deficiency, AOCD most often presents as normocytic normochromic, anaemia. In the present study, the co-morbid illnesses associated in this group of patients were Liver disease, Renal disease, CAHD, GI bleed and Solid organ malignancies.(Table 14). Hence, in elderly patients with microcytic/ normocytic anemia, apart from nutritional iron deficiency, other causes of anemia needs to be determined through endoscopic evaluation of GIT for evidence of blood loss or evaluation for an underlying Renal or Liver disease is necessary. In our study UGI endoscopy done in 29 patients, detected lesions in 82.75% of patients in our study and malignancy in 1 patient. (Table 8). This correlates with Sanchez F et al., (2000)[69] who had shown that UGI lesions were the most frequent cause for iron deficiency.

In the present study, REH of Marrow Micro and Macronormoblastic Erythroid Maturation suggestive of a probable combined B12/ iron deficiency accounted for 15.38% of patients.(Table 13). This is similar (17%) to the observations of Tahlan A et.al., (2008)[70] in their study.

In the present study, Megaloblastic anemia was observed in 20% of patients.(Table 13). This is similar to that shown by pudasini S et al., (2009)[71] who had shown a 18.3% of patients with Megaloblastic anemia.

However Al-Ghazaly J et al., (2006)[72] and Tahlan A et al.loc.cit(2008) showed a high prevalence of Megaloblastic anemia in their studies. In the present study, the patients with Megaloblastic anemia presented with a Dimorphic anemia in 45.45% of cases and Microcytic anemia in 9.09% of cases(Table 17). Seward SJet al.,loc.cit. (1990) in their study have highlighted that a normocytic or even microcytic anemia can be associated in a significant proportion of elderly patients with B12/folate deficiency. In our study Macrocytic anemia was present in 38.46% of patients.(Table 17). Khanduri U et al., (2007)[73] and Mukiibi JM et al., (1992)[74] had shown a higher percentage of macrocytic anemia in patients with Megaloblastic anemia.[87,88] In our study, Pancytopenia was present in 15.38% of patients with Megaloblastic anemia. This relatively correlates with Kumar et al., (2001)[75] who have shown a 20.3% of pancytopenia, whereas Tilak V et al., (1999)[76] and Jha et al., (2008)[77] have shown a higher association of pancytopenia in Megaloblastic anemia.

Malignant hematological disorders comprised 15.38% cases in the present study.(Table 12). This is akin with the observations in the studies by Al-Ghazaly J et al.,loc.cit(2006) and Tahlan A et al.,loc,cit(2008) in which malignant hematological disorders comprised 18.% of cases. Artz et al.,loc.cit(2011) recorded a lower percentage (7.5%) of patients with hematological malignancies. Acute and chronic leukemias accounted for

7.70% of cases in our study.(Table 13). However, Mukiibi JM., et al.,loc.cit (1992) in their study have shown a higher (17.5%) occurrence of Leukemias in elderly.

In the present study, 1(1.54%) patient had a probable diagnosis of AML.(Table 19). Grünewald K et al., (1982)[78] in his study had observed an increased frequency of Acute Myeloid leukemia in older patients with dismal prognosis.

In the present study 3(4.64%) patients were diagnosed with CML, one in chronic phase and two patients in accelerated phase.(Table 20). Similar study by Joosten et al.,(1992)[79] had shown that chronic leukemias accounted for 5% of anemia in elderly.

In anemic elderly, MDS is almost certainly an under diagnosed condition since the evaluation of anemia is less likely to include a bone marrow evaluation in all cases. The prevalence of Myelodysplastic syndrome (MDS) increases in people aged 70 years and older. In the United States, the incidence has been estimated to be 3.5/100,000 to 12.6/100,000 per year[40] .The median age at diagnosis is between 60 and 80years[51]. The diagnosis of the Myelodysplastic syndromes (MDSs) relies on combining the clinical history, morphologic features of the peripheral blood and/or bone marrow sample, and cytogenetic information.

A total of 6 patients were given a diagnosis of MDS (9.23%) in the present study.(Table 13). This is comparable with 5.5% of cases of MDS observed by Joosten et al. (1998)[80] in their study. Marrow iron stores were normal or increased in these patients. The mean age of these patients was 70.5 years. This correlates well with the observations by Mukiibi JM et al.,(1994)[81] who had shown a mean age of 69 years in their study. However Malo JP et al.,(2001)[82] in their study had shown a higher mean age of 78 years in a group of MDS patients. The 6 patients with MDS in the present study included 4 men and 2 women. This is similar to the results of Mukiibi JM et al.,loc.cit.(1994) who had also shown a male preponderance in MDS patients.

Anemia and correlated signs and symptoms are the most relevant, disease-specific manifestation and are the most frequent presenting symptoms of MDS patients. Frequently they can be asymptomatic. In the present study, all patients diagnosed with MDS were symptomatic (100%). However Khalifa MS et al.,(2003)[83] and Malo JP et al.,loc.cit.(2001) had shown that only 81% and 80%of patients respectively were symptomatic. In the present study, Hepatosplenomegaly and skin manifestations were present in 16.67% of the patients. A similar observation has been made by Khalifa MS et al.,loc.cit(2003) who recorded hepato splenomegaly and skin manifestations in 25% of cases.

In the present study, in patients with MDS, co morbid illnesses were present in 66.67% of patients.(Table 14) Wang et al., (2009)[84] had shown a lower (51%) percentage of patients with co morbid disorders.

At presentation, the most common laboratory finding, present in nearly all patients with MDS, is anemia and reticulocytopenia. The anemia is typically Macrocytic but may also be normocytic. Microcytic hypochromic anemia is rarely associated with MDS. In the present study, Anemia associated with MDS was Microcytic in 16.67% of patients. Several series by Barzi A et al.,(2010)[85] Juneja et al.,(1983)[86] Nguyen P et al.,(2009)[87] had shown that Microcytic anemia can be associated with MDS. In the present study, all the MDS patients had severe degree of anemia.(Table 18). Irfan M et al.,(1998)[88] similarly had observed a severe degree of anemia in MDS patients.

Anemia may be accompanied by neutropenia and/or thrombocytopenia, with pancytopenia found in approximately 50% of all cases. Isolated neutropenia or thrombocytopenia is reported in _5% of cases. In the present study Pancytopenia was associated with 50% of MDS patients. A similar (52%) observation has been reported by Chen PH et al., (1992)[89] in their analysis of MDS patients. However studies by Dewulf et al.,loc.cit.(2004)[90] and Irfan M et al.,loc.cit(1998) had shown a lower association of pancytopenia with MDS patients , accounting for

18% and 9% repectively. The higher the degree and number of cell lines decreased, the worse the prognosis.

In the bone marrow, the minimum diagnostic criterion for MDS is dysplasia in _10% of any of the myeloid lineages and less than 20% blast cells of all nucleated cells in the bone marrow unless there are cytogenetic abnormalities suggestive of the diagnosis of MDS as per the World Health Organization (WHO).

The dysplastic features commonly observed in MDS are megaloblastosis, multinuclearity, nuclear budding, intranuclear bridging, karyorrhexis and karyopyknosis and hypolobated megakaryocytes. . In the present study, in MDS patients, Bilineage dysplasia (66.67%) was more common than Unilineage dysplasia (16.67%). This is comparable with the reports of other studies by Dewulf G et al.,loc.cit(2004) and Irfan M et al.,loc.cit.(1998) who had shown a similar observation in MDS patients. However, other studies by Mukiibi JM et al., loc.cit.(1994) and Malo JP et al.,loc.cit(2001) had shown a lower percentage of patients with Unilineage and Bilineage dysplasia. In the present study, MDS with Multilineage dysplasia and Excess Blasts was seen in 16.67% of cases. This is comparable with the results of Mukiibi JM et al.,loc.cit(1994) and Malo JP et al.,loc.cit.(2001) who had shown 21.4% and 24.44% of cases with Refractory anemia with Excess Blasts(RAEB).

However Dewulf et al.,loc.cit.(2004) had encountered only 8% cases in their study.

In the present study five patients, with bone marrow aspiration findings suggesting a possibility of MDS are on follow up studies for further evaluation.

SUMMARY AND CONCLUSION

Anemia in elderly patients should never be regarded as a normal physiological response to aging. The main categories of anemia in older patients are the nutritional anemia attributed to iron deficiency, including blood loss, folate and vitamin B12 deficiency, and Anemia of chronic disease in patients with cancer, infections and other chronic inflammation. MDS is more common in elderly individuals and is also a cause for Unexplained anemia in elderly.

In the present study, we analysed the pattern of Anemia in 65 geriatric patients and found that the anemia was of severe degree in most patients as per WHO criteria. It was often multifactorial. Bone marrow examination done in these patients showed a Reactive Erythroid Hyperplasia of marrow with micronormoblastic maturation suggestive of a probable Iron deficiency in 41.54% of patients. The other less common patterns were Megaloblastic anemia found in 20% patients and Reactive Erythroid Hyperplasia of marrow with micro and macronormoblastic maturation suggestive of a combined B12/iron deficiency in 15.38% of patients. We also encountered 4(7.70%) incidental cases of leukemia in these patients. The present study identified Myelodysplastic syndrome as a cause of Geriatric Anemia in 9.23% of patients.

Thus to conclude, all Elderly patients with Anemia should always be evaluated for an underlying cause. Indiscriminate administration of

iron to a geriatric patient with an unevaluated anemia is not appropriate and may contribute to iron overload especially in AOCD and MDS. Hence a Bone marrow examination is necessary in all cases of geriatric anemia for establishing a diagnosis, particularly in MDS.

BIBLIOGRAPHY

1. Gaskell H, Derry S, Moore A and Henry J et al.. Prevalence of Anaemia in Older Persons: Systematic Review. BMC Geriatrics 2008, 8:1.doi:10.1186/1471-2318-8-1.
2. Balducci L, Ershler WB: Cancer and Ageing: A Nexus at Several Levels. Nat Rev Cancer 2005; 5:655-661.
3. Beghe C, Wilson A, Ershler WB: Prevalence and Outcomes of Anemia in Geriatrics: A Systematic Review of the Literature. Am J Med 2004;116(suppl 7A):3S-10S.
4. Guralnik JM, Eisenstaedt RS, Ferrucci L, et al: Prevalence of Anemia in Persons 65 years and Older in the United States: Evidence for a High Rate of Unexplained Anemia. Blood 2004;104:2263-2268.
5. Bennett, JM, Catovsky, D, Daniel MT, et al. Proposals for the Classification of the Myelodysplastic Syndromes. Br. J. Haem. 1982;51:189–199.
6. Greenberg P, Cox C, LeBeau MM, et al. International Scoring System for Evaluating Prognosis in Myelodysplastic syndromes. Blood. 1997;89:2079-2088.
7. N.P.Zauber; A.G.Zauber. Hematologic Data of Very Old People. Journal of American Medical Association 1987;257(16):2181-2184.

8. United Nations Department of Economic and Social Affairs. Population Division: World Population Prospects. The 2006 Revision. New York: United Nations; 2007.
9. Federal Interagency Forum on Aging-Related Statistics. Older Americans Update 2008: Key Indicators of Well-Being. Washington, DC: US Government Printing Office; 2008.
10. Denny SD, Kuchibhatla MN, Cohen HJ. Impact of Anemia on Mortality, Cognition, and Function in community-dwelling elderly. Am J Med. 2006;119:327–334.
11. Penninx BW, Pahor M, Woodman RC, Guralnik JM. Anemia in Old age is associated with increased Mortality and Hospitalization. J Gerontol A Biol Sci Med Sci. 2006;61:474–479.
12. Marshall A. Lichtman, Thomas J. Kipps, Uri Seligsohn, Kenneth Kaushansky Williams Hematology, 8e, by The McGraw-Hill Companies 2010; 2
13. Beutler E, Drennan W, Block M. The Bonemarrow and Liver in Iron deficiency Anemia:A histopathological study of sections with special reference to stainable iron content. J Lab Clin Med 1954; 43:427.
14. Morrison, S. J., Wandycz, A. M., Akashi, K., Globerson, A et al.The aging of hematopoietic stem cells. Nat Med 1996; 2: 1011–1016.

15. Frikin F, Chesterman C, Pening D, Rush B. De Gruchy's Clinical Hematology in Medical Practice: The Red Cell: Basic Aspect of Anemia. London Blackwell Scientific Pub 1989; 5: 17-36.
16. Robert T. Means Jr. Bertil Glader. Wintrobe's Clinical Hematology 12th Edition. Anemia: General Considerations. Lippincott Williams & Wilkins pub 2009;26: 780-800.
17. Anemia in the Elderly: How Should We Define It, When Does It Matter, and What Can Be Done? Mayo Medical Laboratories.com / communiqué.2008; Vol 33 (2).
18. World Health Organization. Definition of an older or elderly person. http://www.who.int/healthinfo/survey/ageingdefnolder/en/ index.html. Retrieved August 29, 2010
19. Howe R B (1983), Anemia in the elderly. Postgrad. Med 73:153.
20. Demaeyer EM. Preventing and Controlling Iron Deficiency Anaemia through Primary Health Care. World Health Organization, 1989. Reprinted 1990.
21. Kumar, Abbas, Fausto,Aster. Robbins and Cotran Pathologic Basis of Disease, 8/E. Red Blood Cell and Bleeding Disorders. Saunders Elsevier 2010; 14: 639-659.
22. Robert T. Means Jr. Wintrobe's Clinical Hematology 12th Edition. Anemias Secondary to Chronic Disease and Systemic Disorders. Lippincott Williams & Wilkins pub 2009; 45 :1222-23.

23. Elis A, Ravid M, Manor Y, Bental T, Lishner M. “A clinical approach to Idiopathic Normocytic-Normochromic Anemia?” J Am Geriatr Soc 1996; 44:832-4.
24. Price EA, Mehra R, Holmes TH, Schrier SL. Anemia in Older persons: Etiology and Evaluation. Blood Cells Mol Dis. 2011;46(2):159-65.
25. Ganz T. Hepcidin, a key regulator of Iron Metabolism and mediator of Anemia of Inflammation. Blood. 2003;102:783-788.
26. Cesari M, Penninx BW, Lauretani F, Russo CR et al.Hemoglobin levels and Skeletal muscle: results from the InCHIANTI study. J Gerontol A Biol Sci Med Sci. 2004; 59(3):249-54.
27. Bentley DP. Anaemia and chronic disease. Clin Haematol 1982;11:465–479.
28. Kotwal J, Saxena R, Choudhry VP, Dwivedi SN, Bhargava M. Erythrocyte indices for discriminating Thalassaemic and non-thalassaemic microcytosis in Indians. Natl Med J India 1999;12:266-7.
29. Killip S, Bennett JM, Chambers MD. Iron deficiency anemia. Am Fam Physician. 2007;75(5):671-678.
30. Bilic E, Bilic E: Amino acid sequence homology of Thrombopoietin and Erythropoietin may explain thrombocytosis in children with iron deficiency anemia. J Pediatr Hematol Oncol 2003, 25(8):675-676.

31. Hill RS, Pettit JE, Tattersall MH, et al. Iron deficiency and Dyserythropoiesis. Br J Haematol 1972;23:507–512.
32. Carmel R, Green R, Rosenblatt D, et al. Update on cobalamin, folate and homocysteine. In Broudy VC, Prchal JT, Tricot GJ, eds. Hematology 2003 ASH Education Program. 2003:62–81.
33. Erkurt MA, Aydogdu I, Dikilitas M, Kuku I, Kaya E, Bayraktar N, et al. Effects of Cyanocobalamin on Immunity in patients with Pernicious Anemia. Med Princ Pract. 2008;17(2):131-5.
34. Seward SJ, Safran C, Marton KI, Robinson SH (1990) Does the Mean Corpuscular Volume Help Physicians Evaluate Hospitalized Patients with Anemia? J Gen Intern Med 5:187–191
35. Savage DG, Ogundipe A, Allen RH, et al. Etiology and Diagnostic Evaluation of Macrocytosis. Am J Med Sci 2000;319:343–352.
36. Khode K, Marwah S, Buxi G, Yadav RB, Chaturvedi NK. Bone Marrow Examination in cases of Pancytopenia. JIACM 2001;2:55-59
37. Milman N, Schultz-Larsen K. “Iron stores in 70 year old Danish men and women.” Ageing (Milano) 1994; 6(2): 97-103.
38. Ha Thanh Nishino, Chung-Che Chang. Myelodysplastic SyndromesClinico pathologic Features, Pathobiology, and Molecular Pathogenesis. Arch Pathol Lab Med. 2005;129:1299–1310.

39. Mikkael A. Sekeres, Epidemiology, Natural History, and Practice Patterns of Patients with Myelodysplastic Syndromes in 2010. J Natl Compr Canc Netw 2011;9:57-63
40. Parker JE, Fishlock KL, Mijovic A, Czepulkowski B et al. 'Low-risk' Myelodysplastic syndrome is Associated with Excessive Apoptosis and an Increased ratio of Pro- versus Anti-apoptotic bcl-2-related proteins. Br J Haematol.1998;103:1075–1082.
41. Najean Y, Lecompte T. Chronic Pure Thrombocytopenia in Elderly Patients. An aspect of the Myelodysplastic Syndrome. Cancer1989; 64:2506.
42. Bartl R, Frisch B, Baumgart R. Morphologic Classification of the Myelodysplastic Syndromes (MDS): Combined Utilization of Bone Marrow Aspirates and Trephine Biopsies. Leukemia Res 1992; 16:15.
43. Head DR, Kopecky K, Bennett JM, Grenier K et al. Pathogenetic Implications of Internuclear Bridging in Myelodysplastic Syndrome. An Eastern Cooperative Oncology Group/Southwest Oncology Group Cooperative Study.Cancer.1989; 64:2199.
44. Davey FR, Erber WN, Gatter KC, Mason DY: Abnormal Neutrophils in Acute Myeloid Leukemia and Myelodysplastic Syndrome. Hum Pathol 1988;19:454.
45. Kuriyama K, Tomonaga M, Matsuo T, Ginnai I et al. Diagnostic Significance of Detecting Pseudo-Pelger-Hu‘t Anomalies and Micro-

megakaryocytes in Myelodysplastic syndrome. Br J Haematol 1986; 63:665.

46. Coiffier B, Adeleine P, Gentilhomme O, Felman P et al. Myelodysplastic syndromes. A Multiparametric Study of Prognostic Factors in 336 Patients. Cancer.1987; 60:3029,

47. Nimer SD, Golde DW. The 5q- abnormality. Blood. 1987;70:1705–1712.

48. Bowen D, Culligan D, Jowitt S, et al. Guidelines for the Diagnosis and Therapy of Adult Myelodysplastic Syndromes. Br J Haematol. 2003;120:187-200.

49. Kouides PA, Bennett JM. Morphology and Classification of the Myelodysplastic Syndromes and their Pathologic Variants. Semin Hematol.1996;33:95–110.

50. Malcovati L, Della Porta MG, Cazzola M.Predicting survival and leukemic evolution in patients with myelodysplastic syndrome. Haematologica 2006;91:1588–1590.

51. Ferrucci L, Guralnik JM, Bandinelli S, Semba RD et al.Unexplained Anaemia in Older Persons is characterised by Low Erythropoietinand Low Levels of Pro-inflammatory Markers. Br J Haematol 2007;136:849–55.

52. Sasan Makipour, Bindu Kanapuru, and William B. Ershler Unexplained Anemia in the ElderlySemin Hematol. 2008 October; 45(4): 250 254.doi: 10.1053/j.seminhematol.2008.06.003
53. Price EA, Schrier SL. A large proportion of elderly patients with anemia seen in the outpatient setting have unexplained anemia, which is characterized as a Hypoproliferative, Normocytic Anemia. American society of Hematology Annual Meeting; 2007; Atlanta, Georgia. ASH Annual Meeting Poster 366.
54. Max, Does M, Raza A, Mayne ST. Myelodysplastic Syndromes: Incidence and Survival in the United States. Cancer 2007; 109:1536
55. Guralnik JM, Ershler WB, Schrier SL, Picozzi VJ. Anemia in the Elderly: A Public Health Crisis in Hematology. Hematology Am Soc Hematol Educ Program. 2005:528-32.
56. Freedman ML, Sutin DG. Blood disorders and their management in old age. In: Brocklehurst's Textbook of geriatric medicine and gerontology. 5th ed. New York, N.Y.: Churchill Livingstone, 1998:1247–88,
57. Vardiman JW, Harris NL, Brunning RD. The World Health (WHO) classification of the myeloid neoplasms. Blood. 2002;100:2292-2302.
58. Grimwade D, Hills RK. Independent prognostic factors for AML outcome. Hematol 2009: 37:385-95.

59. Shipley JL, Butera JN. Acute myelogenous leukemia. Experimental Hematol 2009; 37: 649-58.

60. Annino L, Crescenzi S, Romani C, Mandelli F. Acute lymphoblastic leukemia in the elderly: results of two different treatment approaches in 49 patients during a 25-year period. Leukemia.1995;9:1643–1647.

61. Brincker H. Population-based age- and sex-specific incidence rates in the 4 main types of leukaemia. Scand J Haematol 1982; 29: 241–249.

62. Amit Bhasin • Medha Y. Rao. Characteristics of Anemia in Elderly: A Hospital Based Study in South India .Indian J Hematol Blood Transfus (Jan-Mar 2011) 27(1):26–32 DOI 10.1007/s12288-011-0056-4.

63. Choi CW, Lee J, Park KH, Choi K et al. Prevalence and Characteristics of Anemia in the Elderly: Cross-Sectional Study of Three Urban Korean Population Samples. American Journal of Hematology 2004; 77:26–30.

64. Petrosyan I, Blaison G et al . Anaemia in the Elderly: An Aetiologic Profile of a Prospective Cohort of 95 Hospitalised Patients.European Journal of Internal Medicine.2012: 23; 6 , 524-528,

65. Ania BJ, Suman VJ, Fairbanks VF. Incidence of Anemia in Older People: An Epidemiologic Study in a Well Defined Population. J Am Geriatr Soc. 1997;45:825–831.

66. Artz AS, Thirman MJ. Unexplained anemia predominates despite an intensive evaluation in a racially diverse cohort of older adults from a referral anemia clinic. J Gerontol A Biol Sci Med Sci. 2011;66(8):925-32.

67. Elzen WP, Willems JM, Westendorp RG.et al., Effect of Anemia and Comorbidity on Functional Status and Mortality in Old Age. CMAJ 2009; 181: 151.

68. Mauro T, Lucca U, Gandini F, Recchia A et al. Prevalence, Incidence and Types of anemia in the Elderly. Haematologica, Vol 95, Issue 11, 1849-1856 doi:10.3324/haematol.2010.023101

69. Sánchez F, Santasuana A, Pañella R, Gómez C, Enciso L et al . Iron deficiency Anemia in Hospitalized Males and Postmenopausal Females. Diagnostic Approach. Gastroenterol Hepatol. 2000 May;23(5):219-23.

70. Tahlan A, Bansal C, Palta A, Chauhan S. Spectrum and Analysis of Bone Marrow Findings in Anemic Cases . Indian J Med Sci 2008;62:336-9.

71. Pudasaini S, Prasad KBR, Rauniyar SK, Shrestha R, Gautam K, Pathak R et al., Interpretation of Bone Marrow Aspiration in Hematological Disorder. Journal of Pathology of Nepal. 2012; 2: 309 -312.

72. Al-Ghazaly J, Al-Selwi AH, Abdullah M, Al-Jahafi AK, Al-Dubai W, Al-Hashdi A. Pattern of Haematological Diseases Diagnosed by Bone Marrow Examination in Yemen: A Developing Country Experience. Clin Lab Haematol 2006;28:376-81.
73. Khanduri U, Sharma A Megaloblastic Anaemia: Prevalence and Causative Factors. Natl Med J India. 2007 Jul-Aug;20(4):172-5.
74. Mukibi JM, Makumbi FA, Gwanzura C. Megaloblastic Anemia in Zimbabwe: Spectrum of Clinical and Hematological Manifestations. East Afr Med J .1992; 9: 83-87.
75. Kumar R, Kalra SP, Kumar H, Anand AC, Madan H. Pancytopenia- A six year Study.JAPI 2001;49:1078-1081
76. Tilak V, Jain R, Pancytopenia- A Clinico- Hematological Analysis of 77 cases. Indian J Pathol Microbiol 1999;42:399-404.
77. Jha A, Sayami G, Adhikari RC, Panta D, Jha R. Bone marrow Examination in cases of Pancytopenia. J Nepal Med Assoc. 2008;47:12-7.
78. Grünewald K, Abbrederis K, Mittermaier P, Huber H. National Cancer Institute (1997) Surveillance, Epidemiology, and End Results (SEER) Program. National Cancer Institute, DCPC, Surveillance Program, Cancer Statistics Branch, Bethesda, MD, USA, 1997.

79. Joosten E, Pelemans W, Hiele M, Noyen J et al. Prevalence and causes of anaemia in a geriatric hospitalized population. Gerontol 1992;38:111–7.

80. Joosten E. Strategies for the Laboratory Diagnosis of some Common Causes of Anaemia in Elderly Patients. Gerontology 2004;50:49–56.

81. Mukiibi JM, Paul B, Gordeuk VR A prospective analysis of 620 bone marrow examinations in Zimbabwe: preliminary observations. Cent Afr J Med. 1989 Jun;35(6):416-9.

82. Tilly-Gentric A, Malo JP, Marion V. Primary Myelodysplasia: Management and Outcome at 3 years in 45 patients age 65 and older. J Am Geriatr Soc 2001;49:1358-1360.

83. Khalifa M, Laatiri MA, Chehata S, Rhaiem K et al. Adult Primary Myelodysplastic syndromes. Report of 36 cases.Tunis Med.2003 Apr;81(4):226-9.

84. Wang R, Gross CP, Halene S, Ma X. Comorbidities and survival in a large cohort of patients with newly diagnosed myelodysplastic syndromes. Leuk Res. 2009;33:1594 –1598

85. Barzi A, Sekeres MA. Myelodysplastic Syndromes: A Practical Approach to Diagnosis and Treatment. Cleve Clin J Med. 2010;77: 37-44.

86. Juneja SK, lmbert M, Jouault H, Scoazec JY, Sigaux F, SultanC. Haematological Features of Primary Myelodysplastic Syndromes at

Initial Presentation: A study of 118 cases. J CIin Pathol1983; 36:29-1135.

87. Nguyen PL. The Myelodysplastic Syndromes. Hematol Oncol Clin North Am. 2009;23:675-691

88. Irfan M, Kakepoto GN, Khursheed M. .Primary myelodysplastic syndrome: clinical spectrum of 53 cases. J Pak Med Assoc. 1998 ;48(3):69-73

89. Chen PH, Kuo CY, Huang CH, Shih LY Primary Myelodysplastic Syndrome: an Analysis Of 56 Patients.Chang Gung Medical Journal.1992;15(3):121-7.

90. Dewulf, Gouin I, Pautas E, Gaussem P, Chaïbi P et al. Myelodysplastic Syndromes Diagnosed in a Geriatric Hospital: Cytological Profile of 100 patients. Epileptic Disorders. 2004; 62(2): 197-202,

APPENDIX - I

Tirunelveli Medical College Hospital

Tirunelveli – 11

Hematology case record

NAME			**LAB NO**	
AGE		**SEX**	**IP NO**	
WARD		**UNIT**		
ADDRESS				
COMPLAINTS				
HISTORY OF PRESENT ILLNESS				
PAST HISTORY	Diabetes Hypertension Acid peptic disease Old PT		Chronic drug ingestion Chemotherapy/ Radiotherapy Renal/Liver Disease Others	
GENERAL EXAMINATION	Build Pallor Icterus Pedal edema Vital signs: Pulse		Lymph nodes Skin ENT Musculoskeletal	

	B.P RR
SYSTEM EXAMINATION	CVS: RS: GIT: RES: CNS:
INVESTIGATIONS	X-ray USG CT MRI

INVESTIGATIONS BASIC LABORATORY CP	
INVESTIGATIONS BASIC LABORATORY BIOCHEMISTRY	
INVESTIGATIONS BASIC LABORATORY OTHERS	
HEMATOLOGY TESTS BONE MARROW ASPIRATION	PROCEDURE : PSIC STERNUM Process : leishman's stain Pearl's stain others
REPORTS PERIPHERAL	

SMEAR STUDY		
BONE MARROW SMEAR STUDY	Cellularity	
	M:E ratio	
	Erythroid series	
	Myeloid series	
	Megakaryocytic series	
	Lymphoid series	
	Monocytic series	
	Others	
	Impression	
	Marrow iron	

DISCUSSION:

FINAL DIAGNOSIS:

APPENDIX II

நோயாளிகளுக்கு அறிவிப்பு மற்றும் ஒப்புதல் படிவம்

ஆய்வு செய்யப்படும் தலைப்பு : **இரத்த சோகை உள்ள முதியவர்களின் எலும்பு மஜ்ஜை ஆய்வு**

நோயாளியின் பெயர் :

நோயாளியின் வயது :

எனக்கு இரத்த சோகை உள்ளது என்பதையும், அதனால் இடுப்பு/ நெஞ்சு எலும்பு மஜ்ஜையை ஊசியின் மூலம் எடுத்து பரிசோதனை செய்வதன் முக்கியத்துவத்தையும் மருத்துவர் மூலம் அறிந்து கொண்டேன்.

என்னுடைய சந்தேகங்களை தீர்க்கவும், அதற்கான தகுந்த விளக்கங்களை பெறவும் வாய்ப்பளிக்கப்பட்டுள்ளது என அறிந்து கொண்டேன். இவ்வாய்வில் நான் தன்னிச்சையாகத்தான் பங்கேற்கிறேன். இந்த ஆய்வுக்காக எலும்பு மஜ்ஜையை எடுக்கும் போதும், எடுத்த பின்னரும் அதன் மூலமாக ஏற்படும் பின் விளைவுகளை (ரத்தப் போக்கு, நோய்க்கிருமி தொற்றுதல்) மருத்துவரின் மூலம் அறிந்து கொண்டு சம்மதம் தெரிவிக்கிறேன்.

இடம்:

தேதி : **நோயாளியின் கையொப்பம்**

APPENDIX III

Staining Procedures

1. Leishman's Stain

Reagents

(i) Leishman powder - 0.15gm

(ii) Methyl Alocohol (acetone free) – 100ml.

Procedure

1. Air dry the smears.
2. Flood the slide with Leishman's stain and allow to stand for 2 minutes.
3. Dilute with double the volume of buffer water and stain for 7- 10 minutes.
4. Wash off the stain in a stream of buffered water until it a pinkish tinge is obtained.
5. Air dry the slides.

2. Reticulocyte Stain

Reagents

Brilliant cresyl blue	–	1.0g.
Phosphate buffer	–	100ml.

Procedure

1. Add 2-3 drops of dye solution in to a small bottle.
2. Add 2-4 volumes of patient's EDTA- anticoagulated blood to the dye solution and mix.
3. Keep the mixture at 37°C for 15 – 20 minutes.
4. Resuspend the cells by gentle mixing and make films in the usual way.

3. Perl's Stain

Reagents

4% potassium ferrocyanide - 25 ml.

4% hydrochloric acid - 25 ml.

Procedure

1. Bone marrow smears are fixed in methanol for 10 minutes.
2. Incubate the smears in freshly prepared acid ferrocyanide solution for 10 -15 minutes.
3. Wash in distilled water.
4. Lightly stain the nuclei with 0.5% aqueous neutral red.

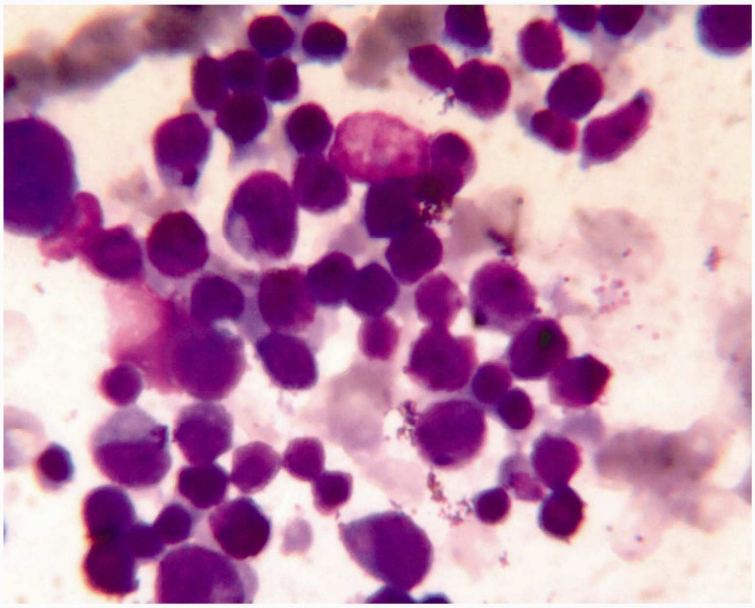


Fig.1. Photomicrograph of a bone marrow smear showing Micronormoblastic erythroid maturation.(Leishman 1000 X).

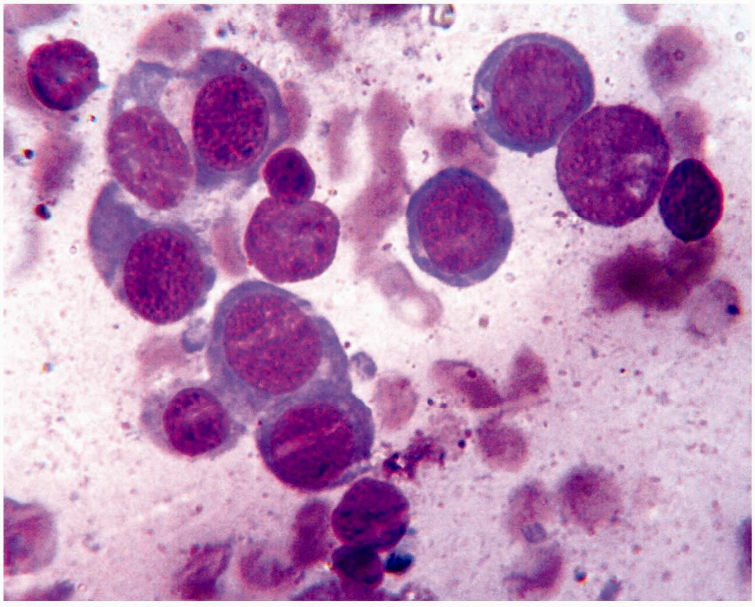


Fig.2. Photomicrograph of a bonemarrow smear showing megaloblastic changes in erythroid and myeloid cells in Megaloblastic anemia. (Leishman 1000 X).

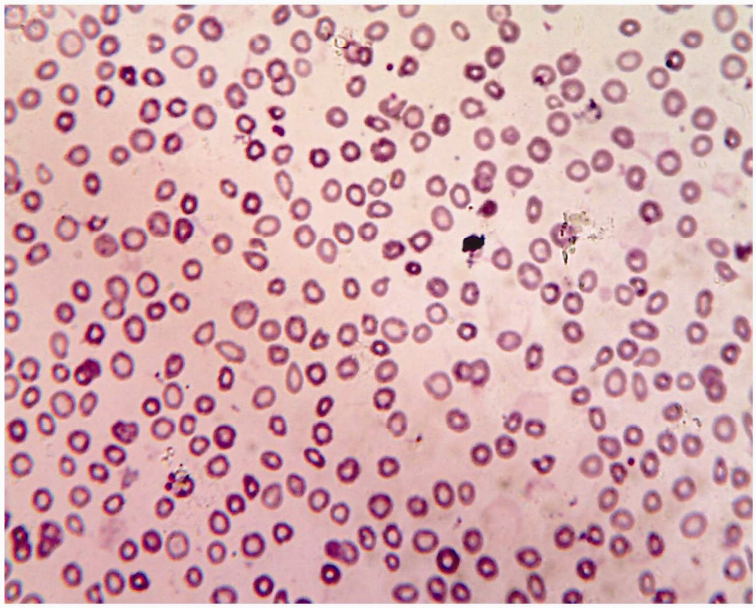


Fig.3. Photomicrograph showing dimorphic anemia in a case of MDS.(Leishman 400 X)

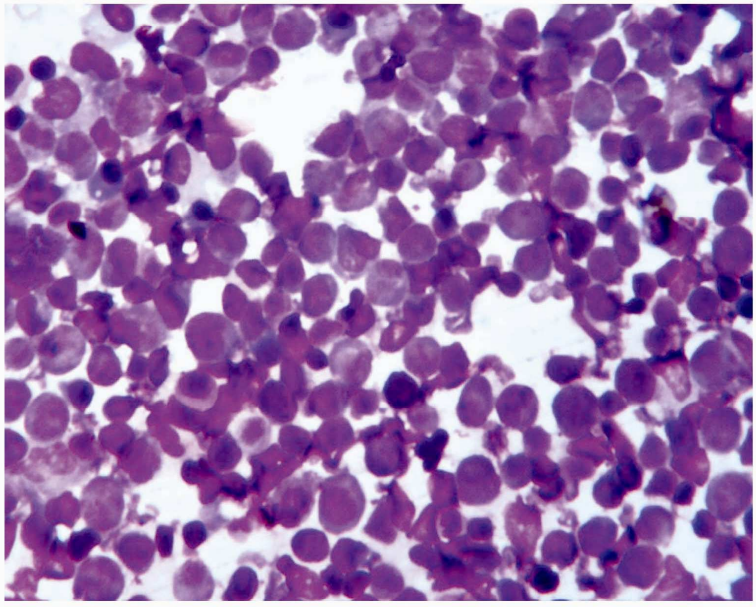


Fig.4. Photomicrograph of a hypercellular marrow in a case of MDS.(Leishman 400 X)

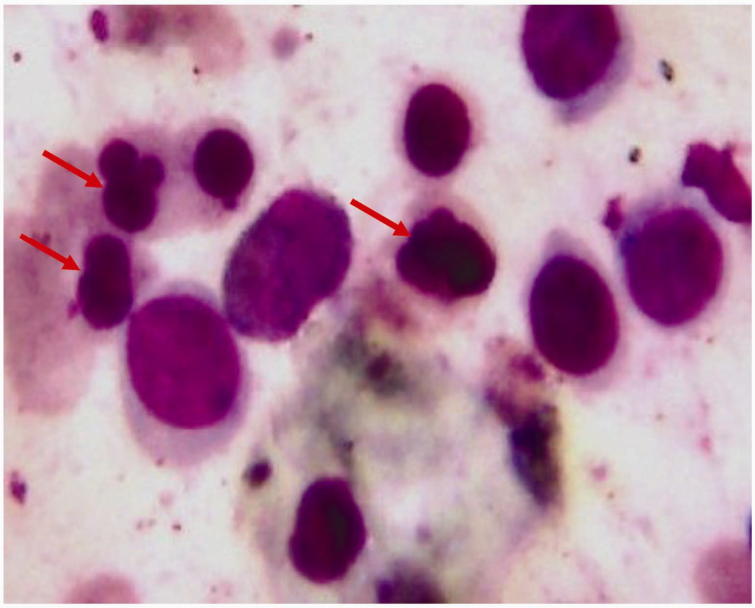


Fig.5. Photomicrograph of a bone marrow smear showing Dyserythropoiesis. (Leishman 400 X)

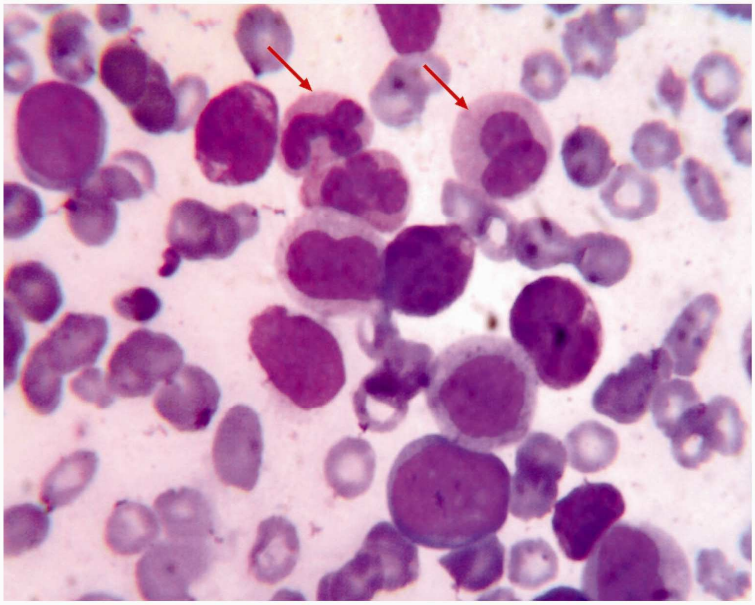


Fig.6. Photomicrograph of a bonemarrow smear showing Dysmyelopoiesis. (Leishman 1000 X)

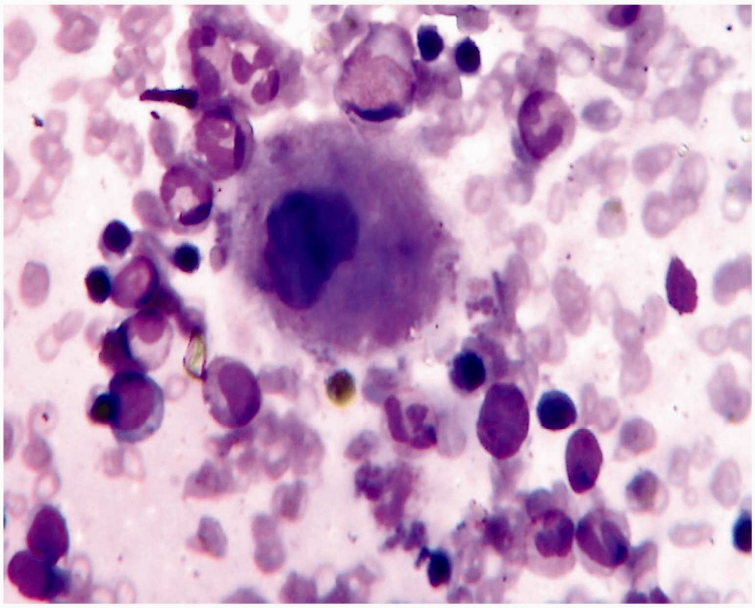


Fig. 7. Photomicrograph of a bone marrow smear showing a Hypolobated Megakaryocyte. (Leishman 400 X)

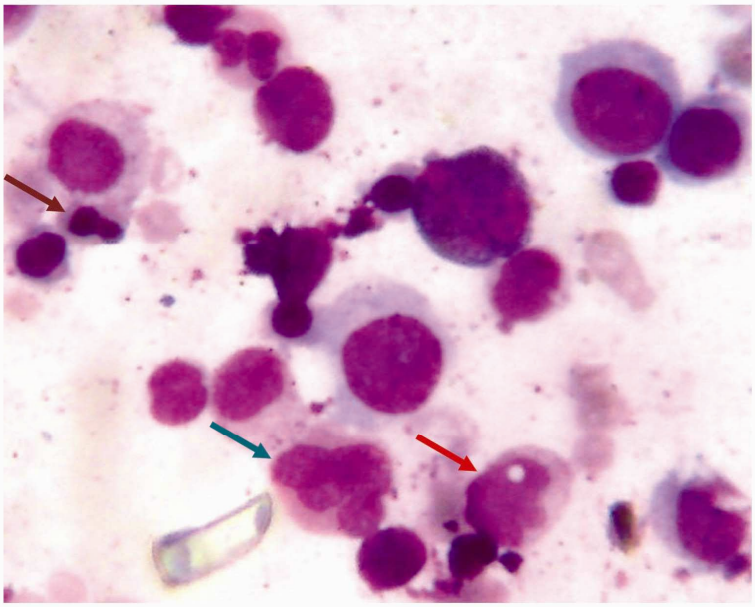


Fig. 8. Photomicrograph of a bone marrow smear showing trilineage dysplasia. (Leishman 400 X)

Brown Arrow - Dyserythropoiesis, Red Arrow - Dysmyelopoiesis, Green Arrow - Dysmorphic Megakaryocyte

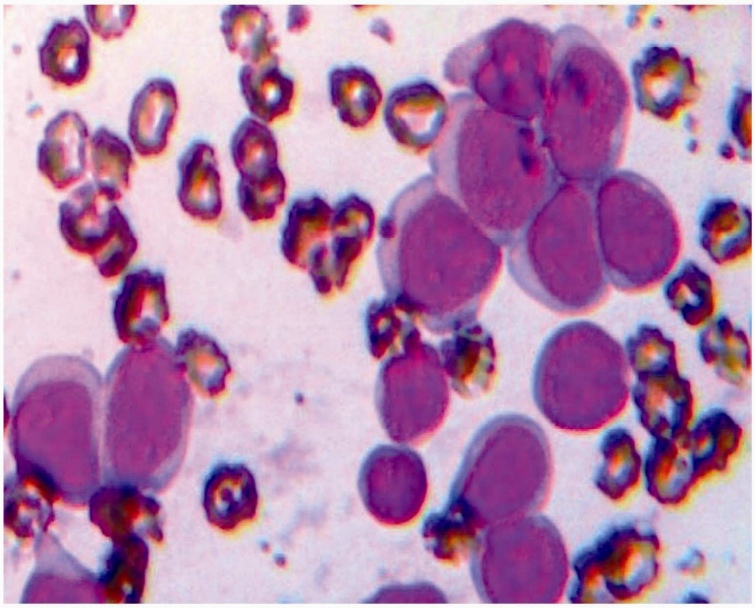


Fig.9. Photomicrograph of a bone marrow smear showing myeloblasts in a case of AML. (Leishman 1000 X)

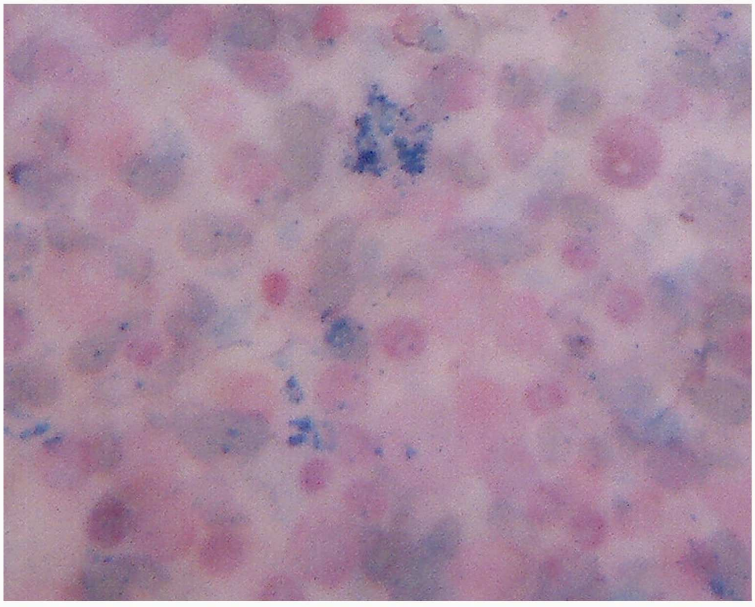


Fig.10. photomicrograph of a bone marrow smear showing increased iron stores. (Perl 400 X)

ID No	Name	Age	sex	Presenting Complaints	Present Co-morbidities 1-DM 2-HT 3-CRD 4-CLD 5-APD 6-Immune Dis 8-GI Bleeds 10-TB 11-Thyroid Dis 12-Cancer 13-GU Bleed 15-Others	Systems	Radiology	Endoscopy	TRBC x106cells/cumm	TLC x103cells/cumm	PLC x105cells/cumm	HB gms/dl	HCT	MCV	MCH	MCHC	Retic C	ESR 1Hr	BSR	BUN	SCR	PSS-RBC Describe RBC	PSS-RBC Impression	PSS-WBC Impression	PSS-PL Impression	Marrow Cellularity 0-Normal 1-Hypo 2-Hyper	Marrow Erythroid Describe	Marrow Myeloid Describe	Marrow Megk Describe	Marrow Impression Describe	marrow iron
1.5654	saraswathy	77	F	dyspnoea, pedal edema decreased urine output	1	mild hepatomegaly, B/L crepts	NAD	normal	3.1	6.2	1.5	6.1	18	102.4	23.2	26.8	0.5	52	35 3	28	1.3	number decreased macrocytic RBCs	macrocytic anemia	Normal	normal	2	number increased megaloblastic maturation	number increased giant metamyelocytes	normal number n morphology	megaloblastic anemia	normal
2.6784	saraswathy	65	F	mass P/V urinary disturbances	1	NAD	NAD	not done	2.5	6.9	2.5	5.6	16	89.3	23.6	27.9	0.3	30 50	106	22	1.1	number decreased normocytic n microcytic RBCs anisopoikilocytosis	dimorphic anemia	RN	reactive thrombocytosis	0	number increased micro n macro normoblastic maturation	incresed in number normal maturation	normal in number n morphology	reactive marrow with micro n macronormoblastic maturation	low
3.1364	seetha	65	F	dyspnoea on exertion	5	hemic murmur	NAD	not done	3	10.5	1.8	6.2	26	74	22	22	1	55 120	148	24	0.8	number decreased microcytic hypochromic RBCs	MCHC anemia anisopoikilocytosis	RN	normal	2	number increased micro n macro normoblasti maturation	normal in number n morphology	normal in number n morphology	REH with micro n macro normoblastic maturation	low
4.4798	esther	62	F	abdominal pain dyspnoea	1,,2. 15	NAD	cardiomegaly mild splenomegaly	mucosal ero	1.82	2.5	30000	4.9	16.7	129.3	41.5	32.1	1	28/50	128	30	1.1	number decreased microcytic hypochromic RBCs	MCHC anemia	leucopeni	thrombocytopenia	2	number decreased micro n macronormoblastic maturation dividing cells dyserythropoiesis	number increased myeloblasts promyelocytes mature forms	variable	MDS multilinege dysplasia	increased
5 .15779	kosalai	65	F	fatigue	1,2,3	hemic murmur, splenomegaly	splenomegaly	not done	2.2	145	0.28	6.9	18.9	85.9	31.5	36.5	. .	.	92	65	3	number decreased microcytic hypochromic RBCs	MCHC anemia	acute leukemia	thrombocytopenia	2	number decreased micronormoblastic maturation	increased in number myeloblasts >90%	decreased	AML-M1	- -
6 .4471	santhivinayagam	65	M	ulcer leg	..	moderate splenomegaly	splenomegaly	not done	2.12	90,000	1.74	8.9	20.9	63.7	21.3	27.4	. .	98 130	70	30	1.1	number decreased microcytic hypochromic RBCs	MCHC anemia	CML - chronic phase	thrombocytosis	2	number decreased micronormoblastic maturation	increase in myeloblasts promyelocytes metamyelocytes	normal number n morphology	CML chronic phase	- -
7 ..9578	bagavathy	66	F	fatigue	5, 8	splenomegaly, tender hepatomegaly	splenomegaly, renal calculi	congested G	2.7	11	0.19	6.4	19.3	72.5	21.3	31.1	1	100 150	104	29	0.9	number decreased microcytic hypochromic RBCs	MCHC anemia	RNL	thrombocytopenia	2	number normal micronormoblastic maturation	increase in metamyelocytes, band forms, mature neutrophils	normal number n morphology	reactive erythroid micronormoblastic maturation	absent
8 .2039	mariyal	63	F	giddiness exertional dyspnoea	1,2,, 6	NAD	fatty liver	not done	2.39	4.3	2.69	6.6	22.1	94	23.8	27.4	0.5	. .	146	38	1.1	number decreased normocytic n macrocytic RBCs	macrocytic anemia	Normal	normal	0	number increased megaloblastic maturation	normal number n maturation	normal number n morphology	megaloblastic anemia	increased
9 ..43923	gnanam	69	F	abdominal pain dyspnoea	6	anemia	…..	….	2.47	9.1	1.02	7.9	36	86.6	24.8	33.2	0.5	99	38	0.7	1.1	number decreased	MCHC anemia	RN	decreased	1	normal number dividing cells	normal	megaloblastic dysmegakaryopoiesis	?MDS	
10 .17294	meenakshi	60	F	giddiness	…	anemia	..	not done	2.67	6.1	3.41	6.9	28.5	74.8	23.6	29.2	1	55	81	21	0.7	number decreased microcytic hypochromic RBCs	MCHC anemia	normal	normal	0	number normal nicronormoblastic maturation	normal number n maturation	normal number n morphology	reactive erythroid with micronormoblastic maturation	absent
11 .17356	santhanam	60	M	exertional dyspnoea	1	submandibular lymph nodes	NAD	not done	2.2	7.8	2.78	7.5	34	83.2	27	28.4	1.4	100	78	22	0.9	number decreased microcytic hypochromic RBCs	MCHC anemia	RN	normal	0	number increased micro n macro normoblastic maturation	normal number n maturation	normal number n morphology	REH with micro n macro normoblastimaturation	normal
12 .32901	selvaraj	62	M	easy fatiguability loss of apetite	, ,	gastric tendern	NAD	erosion	2.9	7.2	1.79	10.4	26.5	88.3	22.3	26.6	1	90	101	28	0.7	number decreased microcytic hypochromic RBCs	MCHC anemia mild anisocytosis	RN	normal	0	number normal micro n macro normoblastic maturation	normal number n maturation	normal number n morphology	REH with micro n macro normoblastic maturation	normal

13 .30148	valliammal	74	F	giddiness, chestpain palpitation	..	anemia	.	not done	2.2	4.1	1.52	7.7	23	86.6	26.1	33.2	0.5	..	64	13	0.5	number decreased normocytic RBCs	normocytic anemia	normal	normal	0	micronormoblastic maturation	normal number n maturation	normal number n morphology	hyperplastic marrow with micronormoblastic maturation	absent
14 .41267	meenambal	73	F	pedal eema chest pain fatigue	15	NAD	NAD	not done	3.3	5.3	2.84	10.8		90.4	43	31.7	0.5	65	96	20	0.8	number decreased normocytic RBCs	normocytic anemia	Normal	normal	0	number normal micro n macro normoblastic maturation	normal maturation	normal number n morphology	REH with micro n macro normoblasti maturation	normal
15 .44321	thangapandi	61	M	giddiness	. .	anemia	NAD	not done	2.6	4.7	3.16	9.3	36	87.4	24.1	29.2	0.5	..	74	16	0.4	number decreased normocytic RBCs	normocytic anemia	RN	normal	0	number increased micronormoblastic maturation	normal number n maturation	normal number n morphology	REH with micro normoblastic maturation	absent
16 .54902	velu	67	F	giddiness loss of apetite fever	, ,	NAD	NAD	not done	1.68	2.9	1.13	5.7	27.9	62.9	24.3	28.6	0.5	60	77	16	0.8	number decreased microcytic hypochromic RBCs	MCHC anemia	normal	normal	0	number increased micronormoblastic maturation	normal number n maturation	normal number n morphology	REH micronormoblastic maturation	absent
17 . 55798	sudalaimuthu	73	M	giddiness	4	hepatomegaly	hepatomegaly	congested G	2.8	7.4	2.53	8.7	28.8	82.6	24.2	29.3	1.2	40	92	14	0.3	number decreased micro n macrocytic RBC	dimorphic anemia	RN	normal	2	number increased micro n macro normoblastic maturation	number increased	number increased normal morphology	REH with micro n macro normoblastic maturation	low
18 .62071	akilandeswari	81	F	loss of apetite vomiting fatigue	..	anemia	NAD	not done	2.9	6.3	1.85	6.8	28.1	81.7	26.3	31.4	1.2	110	81	28	1.1	number decreased anisopoikilocytosis	dimorphic anemia	normal	normal	2	number increased micro n macro normoblastic maturation	number increased	normal number n morphology	REH with micro n macro normoblasti maturation	absent
19 .64721	jeyarani	61	F	fatigue chest pain giddiness	1	DM/ gastritis	NAD	not done	2.06	5.3	2.73	6.3	26.5	59.7	25.3	29.7	0.3	88	92	23	0.7	number decreased microcytic hypochromic RBCs	MCHC anemia	normal	normal	0	number increased micronormoblastic maturation	normal number n maturation	normal number n morphology	reactive erythroid micronormoblastic maturation	absent
20 . 60969	lakshmi	66	F	fatigue diarrho	14	hepatomegaly	hepatomegaly	erosion	1.96	10.6	3.55	5.8	20.1	66.4	21.2	29.6	< 0.5	55 100	137	44	1	number decreased microcytic and macrocytic RBCs	MCHC anemia	normal	reactive thrombocytosis	2	number increased micro n macro normoblastic maturation	normal number n morphology	normal number n morphology	hyperplastic marrow with micronormoblastic maturation	absent
21. 54734	pandaram	69	M	poor apetite giddiness	5	NAD	normal	ulceration	2.47	6.4	5.63	6.9	19.6	79	23.5	29.6	1	50 100	96	25	0.8	number decreased microcytic hypochromic RBCs	MCHC anemia	normal	reactive thrombocytosis	2	number increased micronormoblastic maturation	normal number n morphology	normal number n morphology	REH micronormoblastic maturation	absent
22. 43705	madasamy	70	M	pedal edema dyspnoea	2, 3	der hepatomeg	B/L medical renal disease type III RPD changes	normal	2.68	4.5	2.31	8.2	24.7	99.6	30.6	30.8	0.5	20 130	157	150	5.4	number decreased microcytic and macrocycytic RBCs	dimorphic anemia	RN	normal	2	number increased megaloblastic maturation	increase in metamyelocytes giant band forms	normal in number n morphology	megaloblastic anemia	increased
23 . 45911	madathi	65	F	hemoptysis, fever	. .	hemic murmur, B/L crepts, splenomegaly	old PT, Splenomegaly, increased renal cortical echoes	not done	1.8	125	3.19	7.7	21.3	83.3	26.1	31.3	. .	. .	68	20	0.8	number decreased microcytic hypochromic RBCs	MCHC anemia NRBC	CML-accelerated phase	thrombocytosis	2	number decreased micronormoblastic maturation	increase in myeloblasts promyelocytes metamyelocytes basophils	number increased normal morphology	CML - accelerated phase	- -
24 . 43507	gurusamy	70	M	dyspnoea	3,8,10	systolic murmur B/L crepts	old PT B/L medical renal disease type I RPD changes	mucosal ero	2.5	3.2	0.4	6.8	22.4	128	27	32	1.2	150 165	138	54	2	number decreased macrocytic n normocytic RBCs	dimorphic anemia	leucopenia	thrombocytopenia	1	number increased megaloblastic maturation dividing cells	megaloblastic maturation	normal number n morphology	megaloblastianemia	increased
25 . 61113	shanmugasundaram	62	M	cough palpitation pedal edema	5 , 10	epigatric tenderness hepatomegaly	old PT	not done	2.28	7.8	2.95	5.9	14.4	63.2	14.9	23.6	1.3	10 22	115	34	1.5	number decreased microcytic hypochromic RBCs	MCHC anemia	Normal	normal	2	number increased micronormoblastic maturation	normal number n maturation	normal number n morphology	REH with micro normoblasti maturation	absent
26 . 42094	subbiah	60	M	dyspnoea decreased urine output	2	hemic murmur	cardiomegaly mildsplenomegaly,B/L moderate PE	ulcer, incompetent GEJ	1.36	11.9	3.5	5.4	18	132.4	26.7	30	1	100 130	114	44	1.2	number decreased microcytic hypochromic RBCs	MCHC anemia	reactive neutrophia	thrombocytopenia	2	dividing cells dyserythropoiesis	dysmyelopois is	normal morphology	MDS multilinege dysplasia	increased

27 . 44867	mariappan	62	M	pedal edema facial puffiness	2	hemic murmur	cardiomegaly moderate splenomegaly	not done	1.95	7.3	1.42	5.1	13.8	70.8	16.4	23.2	2	30	60	146	21	0.8	number decreased microcytic hypochromic RBCs	MCHC anemia	normal	normal	2	number increased, micro and macro normoblasti maturation	normal number n morphology	normal number n morphology	REH with micro n macro normoblastimaturation and plasmacytosis	absent
28 . 49305	arumugam	76	M	facial puffiness pedal edema	1,5	b/l crepts	cardiomegaly mild pleural effusion minimal ascitis	congested GI	1.01	5.8	2.92	6.8	16.6	65.3	15.8	24.2	0.5	32	60	98	63	1.2	number decreased microcytic n normocytic	dimorphic anemia	normal	normal	1	micro normoblastic maturation dyserythropoiesis	normal maturation	dysmorphic megks	MDS with plasmacytosis	increased
29. 46728	esakki	72	M	weakness limb	2	stic quadripare	non compressive myelopathy	not done	1.63	1200	1.27	7.7	25.6	104	31.3	30.1	0.5	10	40	135	23	0.8	number decreased microcytic n macrocytic RBCs	macrocytic anemia	leucopeni	normal	1	number decreased megaloblastoid maturation	number decreased dysmyelopoiesis	normal	megaloblastic anemia	normal
30. 50914	esakkkiammal	66	F	mass abdomen	5, 8	liver just palpable massivesplenomegaly	cardiomegaly splenomegaly dilated portal vein	esophageal varices severe PHT/ gastropathy	2.22	4.2	0.41	6.3	18.4	93.6	18.4	22.3	3	26	80	76	16	0.6	number decreased microcytic hypochromic RBCs	dimorphic anemia	normal	thrombocytopenia	0	number increased micronormoblastic maturation	normal in number n morphology	normal in number n morphology	Reactive marrow with micro normoblasti maturation	absent
31 . 40029	veeramariammal	70	F	fever abdominal pain	2,5,	hemic murmur	mild cardiomegaly	congested GI	3.3	12	3.3	9.1	36	89.4	23.6	30.1	<0.5	55	110	86	40	0.8	number decreased microcytic n macrocytic RBCs	dimorphic anemia	reactive neutrophilia	normal	2	number increased megaloblastic maturation	increased giant metamyelocytes band forms	normal number n morphology	megaloblastic anemia	normal
32 . 45982	srinivasan	74	M	giddiness swaying while walking	2	NAD	cerebral atrophy	not done	2.2	3	0.3	6.5	20.1	91	29.4	32.3	1	100	130	65	50	1	number decreased microcytic n normocytic RBCs	dimorphic anemia	leucopeni	thrombocytopenia	1	number decreased dyserythropoiesis	number decreased dysmyelopoiesis	normal number dysmorphic megks	MDS multilinege dysplasia	increased
33 . 47881	paramasivan	62	M	exertional dyspnoea pedal edema	1,2	ESM, hepatosplenomegaly	mild cardiomegaly	congested GI	2.19	4.3	1.35	6.3	20.3	92.7	28.8	31.1	0.5	50	105	117	76	1.4	number decreased microcytic hypochromic RBCs	MCHC anemia	normal	normal	0	number increased micronormoblastic maturation	normal in number n maturation	normal number n morphology	reactive marrow with micro normoblasti maturation s/o IDA	absent
34 . 48220	murugan	63	M	fever	. .	NAD	NAD	not done	3.2	5.8	90	10.4	33.7	97.4	32.9	33.8	1	80	120	78	36	0.9	number decreased normocytic normochromic RBCs	normocytic anemia	reactive neutrophilia	thrombocytopenia	0	number increased normal maturation	number increased normal maturation	normal number n morphology	reactive marrow with micro normoblasti maturation s/o IDA	absent
35 . 32431	poothathan	65	M	abdominal pain dyspnoea	2,.15	splenomegaly	splenomegaly cholelithiasis renal calculi	mucosal erosions	2.26	34.5	1.1	6.5	27.6	72.8	17.8	24.5	1	15	30	60	44	1.2	number decreased normocytic normochromic RBCs occassional macrocyte	normocytic anemia	reactive neutrophilic leucocytosis	normal	2	number normal early megaloblastic maturation	increase in band forms	normal in number n morphology	normocellular marrow with micronormoblastic maturation	absent
36 . 33513	vellathai	65	F	abdominal pain icterus	4,11	ild splenomega	fatty liver mild splenomegaly	congested GI	1.19	11.4	1.73	8.1	24.9	116.9	32.7	31.3	1.5	110	150	106	29	0.8	number decreased normocytic n macrocytic RBCs	macrocytic anemia	reactive neutrophilia	normal	0	number increased megaloblastic maturation	normal number normal maturation	normal number n morphology	megaloblastic anemia	increased
37 . 14794	sundar raj	69	M	giddiness	2, 10	ystolic murmu	NAD	congested GI	2.34	5.3	2.05	6.4	28.1	73.4	24.5	26.2	0.5	50	100	110	28	0.8	number decreased microcytic hypochromic RBCs	MCHC anemia	normal	normal	0	number increased micronormoblastic maturation	normal number normal maturation	normal number n morphology	REH micronormoblastic maturation	absent
38 . 6858	gandhi	85	M	hematemesis	. .	hemic murmur, thyromegaly	B/L MRD type I CKD	stomach - ule	2.1	3.9	2.12	5.6	24	76.4	21.6	28.8	1	50,	105	96	129	2.2	number decreased microcytic and macrocytic RBCs	dimorphic anemia	normal	normal	2	number increased megaloblastic maturation	number increased dysmyelopoiesis	normal in number n morphology	MDS-RA	increased
39 . 9806	ramar	67	M	paraparesis	1, 10	ESM	normal	not done	2.89	3.7	1.93	9.1	31	107.3	31.5	20.4	0.5	12,	45	178	56	1.5	number decreased microcytic n macrocytic RBCs	dimorphic anemia	normal	reactive thrombocytosis	2	number increased micro n macro normoblastic maturation dividing cells	normal in number n morphology	normal number n morphology	? MDS	-

40 . 15364	patrakali	66	F	exertional dyspnoea	. .	ild hepatomega	consolidation lung early type I MRD mild hepatomegaly	congested G	1.27	4	10000	4.7	13.9	90.1	32.2	35.8	0.5	100 150	125	22	1	number decreased microcytic hypochromic RBCs	MCHC anemia	leucopeni	thrombocytopenia	2	number decreased dyserythropoiesis	number increased dysmyelopoiesis	reduced number dysmorphic forms	MDS trilineage dysplasia -RAEB	normal
41. 50536	kanchana	61	F	abdominal pain icterus	1,2,4,6	jaundice hepatomegaly	splenomegaly calculous cholecystitis	congested G	3.2	9.9	4.1	8.7	30.6	72.9	19.6	26.9	1.1	10 22	137	27	0.7	number decreased schistocytes	hemolytic blood picture MCHC	RN	reactive thrombocytosis	0	number increased micronormoblastic maturation	normal number n maturation	normal number n morphology	florid erythroid hyperplasia with micronormoblastic maturation	absent
42 . 47768	muthukrishnan	72	M	giddiness pedal edema	2,3,6	NAD	mild splenomegaly	not done	1.92	2.9	71000	4.8	16	83.3	24.5	29.4	1	45, 85	65	41	1.2	number decreased microcytic n macrocytic RBCs	dimorphic anemia	normal	normal	0	number increased megaloblastic maturation	megaloblastic maturation	normal number n morphology	megaloblastic anemia	normal
43 . 49335	manoharan	65	M	fever, abdominal pain	1,2,3	moderate splenomegaly tender hepatomegaly	hepatosplenomegaly	not done	2.67	7.3	1.56	7.5	23	86.4	28.1	34.6	1.3	100 132	132	95	4.3	number decreased microcytic hypochromic RBCs	MCHC anemia	reactive neutrophilia	normal	0	number increased normal maturation	normal number n maturation	normal number n morphology	erythroid hyperplasiamicronormoblastic maturation	absent
44 . 16214	navaneetham	71	F	exertional dyspnoea vomiting malena	15	venous hum	NAD	erosion	1.96	7.8	2.3	5.9	18.8	78.2	20.5	25.7	2	100 130	126	29	1.1	number decreased microcytic n macrocytic RBCs	dimorphic anemia	normal	normal	2	number increased megaloblastoid maturation dividing cells	number increased	normal in number n morphology	? MDS	low
45 . 42317	ganapathy	66	M	loss of apetite fatigue	. .	moderate splenomegaly	hepatosplenomegaly	not done	1.83	1.53	3.24	6.7	22.1	106	23.2	26.9	. .	60	73	38	1.2	number decreased macrocytic RBCs	macrocytic anemia	CML - acc.phase	reactive thrombocytosis	2	number decreased micro n macronormoblastic maturation	increase in myeloblasts promyelocytes myelocytes band forms mature neutrophils	normal in number n morphology	CML - accc. phase	- -
46 . 4632	rajeshwari	63	F	easy fatiguability loss of apetite	12, 14	ca breast	NAD	not done	2.2	6.5	2.98	10.3	32.3	88.7	25.6	32.1	1.5	50, 100	74	24	0.8	number decreased normoctic normochromic RBCs	normocytic anemia	normal	normal	0	number normal normoblastic maturation	number increased normal maturation	normal number n morphology	erythroid hyperplasia micronormoblastic maturation	absent
47 . 38421	guruvammal	60	F	exertional dyspnoea	12	k/c/o ca breast	NAD	not done	2.91	5.4	3.15	11.1	26.8	97.5	24.3	31.7	1	10 40	90	21	0.8	number decreased normoctic normochromic RBCs	normocytic anemia	normal	normal	1	number increased megaloblastic maturation	dysmyelopois is	normal number n morphology	? MDS	..
48 . 19917	ramani	67	m	loss of apetite	12	/c/o ca alveolu	NAD	not done	2.88	6.5	3.32	10.2	33.1	66.6	26.2	29.7	0.5	55 100	146	20	0.8	number decreased microcytic hypochromic RBCs	MCHC anemia	normal	normal	2	number increased normal maturation	number increased normal maturation	normal number n morphology	REH with micronormoblastic maturation	absent
49 . 37152	lakshmi	60	F	abdominal pain vomiting	8	epigastric mass	antral growth simple hepatic cyst	proliferative growth pylorus erosion	3.1	6000	3.8	5.7	19.4	73.5	22.7	28.5	0.6	40 85	89	32	0.9	number decreased microcytic hypochromic RBCs	MCHC anemia anisopoikilocytosis	normal	normal	0	number increased normal maturation	number increased normal maturation	normal number n morphology	erythroid hyperplasia with micronormoblastic maturation	absent
50. 31693	kannimalar	70	M	giddiness	2, 3, 10	NAD	old PT B/L small kidneys	mucosal ero	2.09	5.4	2.31	7.1	26.4	65.5	16.7	25.1	0.5	42	60	48	1.3	number decreased microcytic n macrocytic RBCs	MCHC anemia	reactive neutrophilia	normal	2	number increased micronormoblastic maturation	normal number n maturation	normal number n morphology	reactive marrow micronormoblastic maturation	absent
51 . 14794	soundarajan	62	M	exertional dyspnoea palpitation	8	hemic murmur, haemorrhoids	NAD	erosion	2.99	6.3	4.32	8.9	30.1	78.9	21.2	32.2	1.2	60	97	20	0.9	number decreased microcytic hypochromic RBCs	MCHC anemia	normal	reactive thrombocytosis	0	number increased micro n macro normoblastic maturation	normal number n maturation	normal number n morphology	REH with micronormoblastic maturation	absent
52 . 3904	velayutham	65	M	chestpain giddiness	. .	NAD	cardiomegaly	not done	2.63	5.9	2.94	9.1	29.6	100.4	22.6	28.3	0.5	50	94	23	0.9	number decreased microcytic n macrocytic RBCs	dimorphic anemia	normal	normal	0	number normal megaloblastic maturation	number normal giant metamyelocytes band forms	normal number n morphology	megaloblastic anemia	increased
53 . 3422	shanmugathammal	65	F	exertional dyspnoea pedal edema	2, 5, 6	ild splenomega	splenomegaly cardiomegaly	normal	1.78	4.7	3.96	7.4	23.5	77.2	19.3	27.4	1	130	112	35	1.1	number decreased microcytic hypochromic RBCs	MCHC anemia	normal	reactive thrombocytosis	0	number increased micronormoblastic maturation	normal number n maturation	normal number n morphology	Reactive marrow with micronormoblastic maturation	absent

54 . 3398	sudalaimadan	61	M	abdominal pain malena	8, 12, 14	/c/o ca stomac	NAD	not done	2.26	5.2	2.13	6.2	24.8	68.7	23.2	25.7	0.5	50	76	28	0.6	number decreased microcytic hypochromic RBCs	MCHC anemia	Normal	normal	0	number increased normal maturation	normal number n maturation	normal number n morphology	REH with micronormoblastic maturation	absent
55 . 4954	krishnan	69	M	giddiness pedal edema	10	hemic murmur	old PT cardiomegaly	not done	1.56	3.2	0.9	6.8	19.6	92.5	18.7	26.6	0.5	40	92	19	1	number decreased macrocytic RBCs	macrocytic anemia	leucopenia	thrombocytopenia	2	number increased megaloblastic maturation	number increased megaloblastic maturation	normal number n morphology	megaloblastic anemia	increased
56 . 3490	jesumichel	65	M	pedal edema loss of weight	2,4	jaundice hepatomegaly	hepatosplenomegaly	erosion	2.99	7.3	2.65	8.3	30.2	56.5	19.3	22.4	1	48	66	22	0.8	number decreased microcytic hypochromic RBCs	MCHC anemia	Normal	normal	0	number increased micro n macronormoblastic maturation	normal number n maturation	normal number n morphology	REH micronormoblastic maturation	absent
57 . 76231	subbulakshmi	61	F	vomiting fever	1	breast on treatn	NAD	not done	3.11	4.8	2.45	7.6	22.3	84.7	24.5	30.2	0.5	110	102	36	0.9	number decreased normocytic BBCs	normocytic anemia NRBC	Normal	normal	0	number increased megaloblastic maturation	normal number n maturation	normal number n morphology	megaloblastic anemia	increased
58 . 50267	ponnammal	66	F	exertional dyspnoea palpitation	15	b/l crepts	pneumonitic changes mild cardiomegaly	not done	1.87	9.2	1.48	5.7	19.4	76.9	21.5	27.3	0.5	55	64	21	0.6	number decreased microcytic hypochromic RBCs	MCHC anemia	reactive neutrophilia	normal	0	number increased micronormoblastic maturation	number increased normal maturation	normal number n morphology	reactive marrow with micro normoblasti maturation	absent
59 . 1400	pathmanaban	61	M	pedal edema abdominal pain	2	NAD	mild splenomegaly	gastric mucosal prolapse	3.47	5	2.98	5.3	22.3	64.3	15.3	23.8	0.5	30 50	112	42	0.9	number decreased microcyti hypochromic RBCs	MCHC anemia	Normal	normal	2	number increased micro normoblastic maturation	normal in number n morphology	normal in number n morphology	REH micronormoblastic maturation	absent
60 . 22754	saroja	68	F	pedal edema facial puffiness	15	ystolic murmu	cardiomegaly B/L minimal PE	not done	2.23	6.4	2.11	8.3	20.6	98.4	22.1	28.4	0.5	100	75	41	1.1	number decreased microcytic n macrocytic RBCs	dimorphic anemia	Normal	normal	2	number increased megaloblastic maturation	number increased megaloblastic maturation	normal number n morphology	megaloblastic anemia	low
61 . 14865	mangaiyarkarasi	63	F	chest pain, exertional dyspnoea				not done	1.88	6.5	3.26	8.3	36.8	86.5	22.6	28.7	0.5	..	113	43	0.6	number decreased microcytic hypochromic	MCHC anemia	Normal	normal	0	micro normoblastic maturation	normal	normal	REH with micro n macro normoblasti maturation	
62 . 33871	parvathy	66	F	fatigue	3	NAD	NAD	not done	2.93	5.6	2.29	8.2	31.6	69.6	25.3	30.4	0.6	90	89	23	0.6	number decreased	MCHC anemia	normal	reactive thrombocytosis	0	number increased micronormoblastic maturation	normal number n maturation	normal number n morphology	hyperplastic marrow with micronormoblastic maturation	absent
63 . 6297	muthulakshmi	71	F	fatigue vomitir	. .	gastric tendern	NAD	mucosal eros	3.06	3.8	1.06	11.2	33.6	77.6	25.2	31.7	0.5	100	95	26	0.7	number decreased	normocytic anemia	normal	normal	0	micro normoblastic maturation	normal	normal	reactive hyperplasia micronormoblastic maturation	absent
64. 14709	sagunthala	76	F	fatigue, cough, constipation	….	anemia	….	…..	2.3	8.6	2.64	6.6	32	72.3	22.7	31.1	0.5	63	20	1	..	.	MCHC anemia	RN	normal	1	megaloblastic Yserythropoiesis	normal	normal	? MDS	
65 . 12487	subramanian	65	M	dyspnoea chestpain	2	NAD	cardiomegaly	normal	3	4.5	4.22	7	24	66	18.7	22.6	<0.5	5, 20	75	16	1.3	number decreased macrocytic RBCs	macrocytic anemia	Normal	normal	2	number increased megaloblastic maturation	number increased myelocyte metamyelocyte giant band forms	normal in number n morphology	megaloblastic anemia	increased

1.5654	saraswathy	77	F	dyspnoea, pedal edema decreased urine output	1	7
10.2039	mariyal	63	F	giddiness exertional dyspnoea	1,2,10, 6	3, 7
13. 43705	madasamy	70	M	pedal edema dyspnoea	2, 3	7
15. 43507	gurusamy	70	M	dyspnoea	3,8,10,15	7
22. 40029	veeramariamn	70	F	fever abdominal pain	2,5,7	7
27. 33513	vellathai	65	F	abdominal pain icterus	4,11	7
33. 47768	muthukrishna	72	M	giddiness pedal edema	2,3,6	7
43. 3904	velayutham	65	M	chestpain giddiness	15	7
46. 4954	krishnan	69	M	giddiness pedal edema	5, 10, 15	7
50. 22754	saroja	68	F	pedal edema facial puffiness	15	7

					2, 15	
7. 12487	subramanian	65	M	dyspnoea chestpain		7

2	0	3	0	0	0	1	1	0
2	0	1	0	0	0	0	1	0
2	3	2	0	1	0	1	1	0
2	3	3	0	0	0	0	1	0
2	3	1	0	0	0	0	0	0
2	0	2	0	1	1	0	0	0
2	2,3	1	0	1	0	0	1	0
2	2,3	1	0	0	0	0	1	0
2	3	3	0	0	0	0	1	0
2	3	1	0	0	0	0	1	0

2	2,3	3	0	0	0	0	0	0

0	76	110/70	22	mild hepatomegaly, B/L crepts	NAD	pale upperGIr
0	66	130/100	26	NAD	fatty liver	normal
0	78	160/100	23	der hepatomegaly	B/L medical renal disease type III RPD changes	normal
0	85	120/ 70	18	systolic murmur B/L crepts	old PT B/L medical renal disease type I RPD changes	pale upper GI
4	68	130/90	24	hemic murmur	mild cardiomegaly	normal
0	71	140/70	19	ild splenomegaly	fatty liver mild splenomegaly	congested GI
0	93	140/70	22	NAD	mild splenomegaly	normal
0	88	120/70	22	NAD	cardiomegaly	normal
0	68	140/90	20	hemic murmur	old PT cardiomegaly	pale UGI muc
0	80	150/70	14	ystolic murmur	cardiomegaly B/L minimal PE	pale UGI muc

0	84	130/70	20	NAD	cardiomegaly	normal

3.1	6.2	1.5	6.1	18	102.4	23.2	26.8
2.39	4.3	2.69	6.6	22.1	94	23.8	27.4
2.68	4.5	2.31	8.2	24.7	99.6	30.6	30.8
2.5	3.2	0.4	6.8	22.4	128	27	32
3.3	12	3.3	9.1	36	89.4	23.6	30.1
1.19	11.4	1.73	8.1	24.9	116.9	32.7	31.3
1.92	2.9	71000	4.8	16	83.3	24.5	29.4
2.63	5.9	2.94	9.1	29.6	100.4	22.6	28.3
1.56	3.2	0.9	6.8	19.6	92.5	18.7	26.6
2.23	6.4	2.11	8.3	20.6	98.4	22.1	28.4

3	4.5	4.22	7	24	56	18.7	22.6

0.5	52	35 3	28	1.3	0.6	18	22	86
0.5	50 130	146	38	1.1	0.6	18	29	107
0.5	20 130	157	150	5.4	1.1	46	39	114
0.5	150 165	138	54	2	0.7	22	28	101
<0.5	55 110	86	40	0.8	0.9	21	32	97
1.5	110 150	106	29	0.8	0.9	32	21	96
0.5	45, 85	65	41	1.2	3.8	48	24	65
0.5	50	94	23	0.9	0.6	34	36	102
0.5	40	92	19	1	1.1	16	21	76
0.5	100	75	41	1.1	1.2	37	44	107

0.5	5, 20	75	16	1.3	0.5	18	21	88

5.8	3.1	1.1	83	0.7	number decreased macrocytic RBCs	macrocytic anemia	3	normal number n morphology
5.3	2.2	1.1	86	0.7	number decreased normocytic n macrocytic RBCs	macrocytic anemia	3	normal number n morphology
4.8	2.8	0.7	74	1.9	number decreased microcytic and macrocycytic RBCs	dimorphic anemia	4	increase in neutrophils
5.6	3	1.3	88	0.3	number decreased macrocytic n normocytic RBCs	dimorphic anemia	4	number decreased
6.2	2.9	1.9	52	3.5	number decreased microcytic n macrocytic RBCs	dimorphic anemia	4	number increased shift to left
6.2	3.2	68	4.5	2.1	number decreased normocytic n macrocytic RBCs	macrocytic anemia	3	increase in neutrophils
6.7	3.2	1.2	89	2.3	number decreased microcytic n macrocytic RBCs	dimorphic anemia	4	normal in number n morphology
5.4	3	1.2	66	0.4	number decreased microcytic n macrocytic RBCs	dimorphic anemia	4	normal number n morphology
6.2	3.1	1.2	65	0.7	number decreased macrocytic RBCs	macrocytic anemia	3	number decreased
5.6	3.2	0.5	79	0.4	number decreased microcytic n macrocytic RBCs	dimorphic anemia	4	normal number n morphology

5.9	2.7	1.3	78	0.3	number decreased macrocytic RBCs	macrocytic anemia	3	normal in number n morphology

Normal	0	normal number n morphology	normal	0	0	number increased megaloblastic maturation	number increased giant metamyelocytes	normal number n morphology
Normal	0	normal number n morphology	normal	0	0	number increased megaloblastic maturation	normal number n maturation	normal number n morphology
RN	1	normal number and morphology	normal	0	0	number increased megaloblastic maturation	increase in metamyelocytes giant band forms	normal in number n morphology
leucopenia		number reduced giant platelets	thrombocytopenia	3	0	number increased megaloblastic maturation dividing cells	megaloblastic maturation	normal number n morphology
reactive neutrophilia	1	normal number n morphology	normal	0	0	number increased megaloblastic maturation	increased giant metamyelocytes band forms	normal number n morphology
reactive neutrophilia	1	normal number n morphology	normal	0	0	number increased megaloblastic maturation	normal number normal maturation	normal number n morphology
normal		normal in number n morphology	normal	0	0	number increased megaloblastic maturation	megaloblastic maturation	normal number n morphology
normal	0	normal number n morphology	normal	0	0	number normal megaloblastic maturation	number normal giant metamyelocytes band forms	normal number n morphology
leucopenia		number decreased normal morphology	thrombocytopenia	2	0	number increased megaloblastic maturation	number increased megaloblastic maturation	normal number n morphology
Normal	0	normal number n morphology	normal	0	0	number increased megaloblastic maturation	number increased megaloblastic maturation	normal number n morphology

Normal	0	normal in number n morphology	normal	1	0	number increased megaloblastic maturation	number increased myelocyte metamyelocyte giant band forms	normal in number n morphology

megaloblasti
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					1,2, 15	
4.4798	esther	62	F	abdominal pain dyspnoea		7
					3,8,10,15	
15. 43507	gurusamy	70	M	dyspnoea		7
					2, 15	
17. 42094	subbiah	60	M	dyspnoea decreased urine output		7
					1,5,15	
19. 49305	arumugam	76	M	facial puffiness pedal edema		7
					2	
20. 46728	esakki	72	M	weakness limbs		7
					2, 10, 15	
23. 45982	srinivasan	74	M	giddiness swaying while walking		7
					8	
29. 6858	gandhi	85	M	hematemesis		7
					4, 15	
31. 15364	patrakali	66	F	exertional dyspnoea		7

2	0	3	0	0	1	0	0	0
2	3	3	0	0	0	0	1	0
2	1,2,3	3	0	0	0	0	1	0
2	1,2,3	1	0	0	0	0	1	0
2	1, 2, 3	2	0	0	0	0	1	0
2	2,3	3	0	0	0	0	1	0
2	3	3	0	0	0	0	0	0
2	3	3	0	0	0	1	1	0

0	86	140/80	20	NAD	cardiomegaly splenomegaly	normal
0	85	120/ 70	18	systolic murmur B/L crepts	old PT B/L medical renal disease type I RPD changes	pale upper GI
0	94	160/90	22	hemic murmur	cardiomegaly mildsplenomegaly,B/L moderate PE	ulcer, incompetent GEJ
0	76	140/70	16	b/l crepts	cardiomegaly mild pleural effusion minimal ascitis	normal
0	80	150/80	18	stic quadriparesis	non compressive myelopathy	pale UGI muc
0	74	120/80	16	NAD	cerebral atrophy	normal
0	70	11o /70	23	hemic murmur, thyromegaly	B/L MRD type I CKD	normal
1, 2	100	110/70	20	ld hepatomegaly	consolidation lung early type I MRD mild hepatomegaly	pale UGI muc

1.82	2.5	30000	4.9	16.7	129.3	41.5	32.1
2.5	3.2	0.4	6.8	22.4	128	27	32
1.36	11.9	3.5	5.4	18	132.4	26.7	30
1.01	5.8	2.92	6.8	16.6	65.3	15.8	24.2
1.63	1200	1.27	7.7	25.6	104	31.3	30.1
2.2	3	0.3	6.5	20.1	91	29.4	32.3
2.1	3.9	2.12	5.6	24	76.4	21.6	28.8
1.27	4	10000	4.7	13.9	90.1	32.2	35.8

1	28/50	128	30	1.1	2.1	21	28	90
0.5	150 165	138	54	2	0.7	22	28	101
1	100 130	114	44	1.2	2.4	19	22	85
0.5	32 60	98	63	1.2	2.7	105	123	197
0.5	10 40	135	23	0.8	1.1	62	28	84
1	100 130	65	50	1	1	38	27	79
1	50, 105	96	129	2.2	1.2	36	26	206
0.5	100 150	125	22	1	0.6	31	31	85

4.6	2.5	1.3	103	1.1	number decreased microcytic hypochromic RBCs	MCHC anemia	2	number decreased
5.6	3	1.3	88	0.3	number decreased macrocytic n normocytic RBCs	dimorphic anemia	4	number decreased
5.3	2.2	1.7	89	1	number decreased microcytic hypochromic RBCs	MCHC anemia	2	increase in neutrophils toxic vacuoles
5.8	3	1.5	61	0.5	number decreased microcytic n normocytic	dimorphic anemia	4	normal in number n morphology
6	2.7	1.6	81	1	number decreased microcytic n macrocytic RBCs	macrocytic anemia	3	number decreased
6.3	3.2	2	92	3.3	number decreased microcytic n normocytic RBCs	dimorphic anemia	4	number decreased
5.3	2.6	1.3	86	0.6	number decreased microcytic and macrocytic RBCs	dimorphic anemia	4	normal in number n morphology
7.9	4.4	1.2	89	1	number decreased microcytic hypochromic RBCs	MCHC anemia	2	number decreased

leucopenia		number reduced normal morphology	thrombocytopenia	2	2	number decreased micro n macronormoblastic maturation dividing cells dyserythropoiesis	number increased myeloblasts promyelocytes mature forms	variable
leucopenia		number reduced giant platelets	thrombocytopenia	3	0	number increased megaloblastic maturation dividing cells	megaloblastic maturation	normal number n morphology
reactive neutrophia	1	number reduced normal morphology	thrombocytopenia	2	2	dividing cells dyserythropoiesis	dysmyelopoisis	normal morphology
normal	0	normal in number n morphology	normal	0	1	dyserythropoiesis	normal maturation	dysmorphic megks
leukopenia		normal number n morphology	normal	0	1	number decreased megaloblastoid maturation	number decreased dysmyelopoiesis	normal
leucopenia		number decreased normal morphology	thrombocytopenia	3	1	number decreased dyserythropoiesis	number decreased dysmyelopoiesis	normal number dysmorphic megks
normal	0	normal in number n morphology	normal	0	2	number increased megaloblastic maturation	number increased dysmyelopoiesis	normal in number n morphology
leucopenia		number decreased	thrombocytopenia	2	2	number decreased dyserythropoiesis	number increased dysmyelopoiesis	reduced number dysmorphic forms

	MDS RAEB
	megaloblastic dyserythropoiesis
	MDS
plasmacytosis	MDS
	MDS
	MDS
	MDS
	MDS trilineage dysplasia - RAEB

AGE		GENDER		PRESEN
				d
60 - 69 yrs	39	females -	22	
				pe
70 - 79 yrs	10	males -	28	
>80 yrs	1			ab
				n
				bl

CO- MORBID CONITIONS

DM - 11
HT - 19
liver - 6
renal - 6
PT - 10

APD - 10
CA - 5
thyroid - 1
bleeding - 7
immune - 4

NTING COMPLAINTS	DIET	PALLOR	SPLEEN	PEDAL EEMA
lyspnoea - 16	veg - 5	sclera -22	no - 36	yes - 32
fatigue - 6	non veg - 45	tongue - 11	mild - 7	no - 18
edal edema - 12		both - 17	moderate - 5	
dominal pain - 6	HABITS		marked - 2	
giddiness - 8	no - 12	L.N		
ieuro. Sym - 3	alcohol - 7	no - 41	LIVER	
leeding sym - 3	smoking - 16	isolated - 9	no - 38	
others - 8	tea - 30		mild - 10	
			moderate - 2	

anemia severity

mild - 3

moderate- 18

severe - 29

PERIPHERAL SMEAR			BONE MARROW
RBC	WBC	PLATELETS	CELLULARITY
normocytic - 5	normal - 29	normal - 30	normo -36
microcytic - 25	reactive - 11	high - 10	hypo - 4
macrocytic - 7	leucopenia -6	low - 4	hyper - 10
dimorphic - 13	leukemia - 4		
	acute - 1		
	chronic - 3		
pancytopenia - 5			

neoplasms - 10

non neoplastic - 40

AML - 1

CML - 3

- chronic phase - 2
- accelerate phase - 1

MDS - 6

- RA- 1
- RAEB- 2
- RCMD- 3

megaloblastic anemia - 14

- meg. dYsmye elopoiesis - 1
- meg. dYserythropoiesis - 1

reactive marrow - 26

hyperplastic - 10

IDA - 11

combined - 3

possibility of MDS - 2

p/Omds

					15		
35. 16214	navaneetham	71	F	exertional dyspnoea		7	1
					2		
20. 46728	esakki	72	M	weakness limbs		7	2
					1, 10		
30. 9806	ramar	67	M	paraparesis		7	2
					3,8,10,15		
15. 43507	gurusamy	70	M	dyspnoea		7	2
					12		
38. 38421	guruvammal	60	F	exertional dyspnoea		2	2
61	gnanam	69	F				
52		76	F				

0	3	0	0	0	0	1	0	0
1, 2, 3	2	0	0	0	0	1	0	0
2,3	1	0	0	0	0	1	0	0
3	3	0	0	0	0	1	0	0
0	1	0	0	0	0	0	0	0

68	110/ 80	14	venous hum	NAD	normal	1.96
80	150/80	18	stic quadriparesis	non compressive myelopathy	pale UGI muc	1.63
88	120/80	14	ESM	normal	normal	2.89
85	120/ 70	18	systolic murmur B/L crepts	old PT B/L medical renal disease type I RPD changes	pale upper GI	2.5
64	130/ 80	14	ca breast	NAD	normal	2.91

7.8	2.3	5.9	18.8	78.2	20.5	25.7	2
1200	1.27	7.7	25.6	104	31.3	30.1	0.5
3.7	1.93	9.1	31	107.3	31.5	20.4	0.5
3.2	0.4	6.8	22.4	128	27	32	1.2
5.4	3.15	11.1	26.8	97.5	24.3	31.7	1

100 130	126	29	1.1	0.9	32	40	112	6
10 40	135	23	0.8	1.1	62	28	84	6
12, 45	178	56	1.5	0.6	46	37	99	7.5
150 165	138	54	2	0.7	22	28	101	5.6
10 40	90	21	0.8	0.8	24	21	108	6.2

2.9	1.2	69.1	0.6	number decreased microcytic n macrocytic RBCs	dimorphic anemia	4	number normal hypersegmented neutrophils	normal
2.7	1.6	81	1	number decreased microcytic n macrocytic RBCs	macrocytic anemia	3	number decreased	leukopenia
3.5	0.6	60	0.5	number decreased microcytic n macrocytic RBCs	dimorphic anemia	4	normal in number n morphology	normal
3	1.3	88	0.3	number decreased macrocytic n normocytic RBCs	dimorphic anemia	4	number decreased	leucopenia
3.1	1.8	92	0.6	number decreased normoctic normochromic RBCs	normocytic anemia	1	normal number n morphology	normal

0	normal in number n morphology	normal	0	0	number increased micro n macronormoblastic maturation dividing cells	number increased dysmyelopoiesis	normal in number n morphology
	normal number n morphology	normal	0	1	number decreased megaloblastoid maturation	number decreased dysmyelopoiesis	normal
0	number increased giant platelets	reactive thrombocytosis	3	0	number increased micro n macro normoblastic maturation dividing cells	normal in number n morphology	normal number n morphology
	number reduced giant platelets	thrombocytopenia	3	0	number increased megaloblastic maturation dividing cells	megaloblastic maturation	normal number n morphology
0	normal number n morphology	normal	0	1	number increased megaloblastic maturation	dysmyelopoiesis	normal number n morphology

REH with micro n macro normoblasti maturation with features s/o myelodyspla sia	p/o MDS
p/o MDS	p/o MDS dyserythropo iesis
reactive marrow with micro n macronormo blastic maturationpo ssibility of MDSmay be thought of	p/o MDS dyserythropo iesis
megaloblasti c dyserythropo iesis	p/o MDS meg dyserythro
megaloblasti c ysmyelopoie sis	p/o MDS meg dysmyelo
megaloblasti c dysmegakary opoiesis	
meg. Yserythropoi esis	meg. Yserythropoi esis

ID No	Name	Age	Gender	Presenting Complaints	Code	Present Co-morbidities 1-DM 2-HT 3-Chronic Renal Dis 4-Chr Liver Dis 5-APD 6-Chr Immune Dis 7-Menstrual Dis 8-Chr GI Bleeds 9-Splenic Enlargments 10-TB 11-Thyroid Dis 12-Cancer 13-GU Bleeding 14-Diarrhoea 15-Others	Treatment for Co-Morbidities 1-RT 2-CT 3-Steroids 4-AntiThyroid 5-Hemodialysis 6-OCD 7-Others	Dietary History 1-Vegan 2-Non Vegan
6.15779	kosalai	65	F	fatigue		1,2,3	7	2
36. 42317	ganapathy	66	F	loss of apetite fatigue		15	7	2
8.4471	santhivinayag	65	M	ulcer leg		10	7	2
14. 45911	madathi	65	F	hemoptysis, fever		15	7	2

33.	47768	muthukrishna	72	M	giddiness pedal edema	2,3,6	7	2
43.	3904	velayutham	65	M	chestpain giddiness	15	7	2
	10.2039	mariyal	63	F	giddiness exertional dyspnoea	1,2,10, 6	3, 7	2
48.	76231	subbulakshmi	61	F	vomiting fever	1, 10	1, 2	2
27.	33513	vellathai	65	F	abdominal pain icterus	4,11	7	2
50.	22754	saroja	68	F	pedal edema facial puffiness	15	7	2
	1.5654	saraswathy	77	F	dyspnoea, pedal edema decreased urine output	1	7	2
7.	12487	subramanian	65	M	dyspnoea chestpain	2, 15	7	2
22.	40029	veeramariamr	70	F	fever abdominal pain	2,5,7	7	2
13.	43705	madasamy	70	M	pedal edema dyspnoea	2, 3	7	2

46.	4954	krishnan	69	M	giddiness pedal edema	5, 10, 15	7	2
19.	49305	arumugam	76	M	facial puffiness pedal edema	1,5,15	7	2
23.	45982	srinivasan	74	M	giddiness swaying while walking	2, 10, 15	7	2
29.	6858	gandhi	85	M	hematemesis	8	7	2
31.	15364	patrakali	66	F	exertional dyspnoea	4, 15	7	2
	4.4798	esther	62	F	abdominal pain dyspnoea	1,2, 15	7	2
17.	42094	subbiah	60	M	dyspnoea decreased urine output	2, 15	7	2
	66		63	F				
	60		73	F				
	63		60	M				
	67	selvaraj	62	M				
	56		61	M				

					15		
2.6784	saraswathy	65	F	mass P/V urinary disturbances		7	2
					4		
18. 44867	mariappan	62	M	pedal edema facial puffiness		7	2
57	sudalaimuthu	73	M				
					5		
3.1364	seetha	65	F	dyspnoea on exertion		7	2
					5, 12		
12. 54734	pandaram	69	M	poor apetite giddiness		7	2

Habits 0-No 1-Alcohol 2-Smoking 3-Tea/Cofee excess intake	Palor 1-Sclrea 2-Tongue 3- Both	Cyanosis 0-No 1-Yes 2-Marked	Lymphad enopathy 0-No 1-Isolated 2-Generalized	Spleen 0-No 1-Minimal 2-Moderate 3-Marked	Liver 0-No 1-Mild 2-Moderate 3-Marked	Pedal Edema 0-No 1-Yes	Clubbing 0-No 1-Yes	Skin Changes 0-No 1-Petichiae 2-Purpura 3-Hyperpigmentation 4-Hypopigmentation 5-Sclerosis
0	3	0	0	2	0	0	0	0
2	1	0	1	2	0	1	0	0
1, 3	2	0	0	3	0	1	0	3
3	1	0	0	2	0	1	0	0

2,3	1	0	1	0	0	1	0	0
2,3	1	0	0	0	0	1	0	0
0	1	0	0	0	0	1	0	0
0	2	0	0	0	0	0	0	0
0	2	0	1	1	0	0	0	0
3	1	0	0	0	0	1	0	0
0	3	0	0	0	1	1	0	0
2,3	3	0	0	0	0	0	0	0
3	1	0	0	0	0	0	0	4
3	2	0	1	0	1	1	0	0

3	3	0	0	0	0	1	0	0
1,2,3	1	0	0	0	0	1	0	0
2,3	3	0	0	0	0	1	0	0
3	3	0	0	0	0	0	0	0
3	3	0	0	0	1	1	0	1, 2
0	3	0	0	1	0	0	0	0
1,2,3	3	0	0	0	0	1	0	0

3	3	0	0	0	0	0	0	3
3	1	0	0	0	1	1	0	4
3	1	0	0	0	0	0	0	0
2,3	2	0	0	0	0	1	0	0

Peripheral Pulses	BP	Resp Rate	Systems Describe Changes	Code Changes	Radiology Describe Changes	Code Changes	Endoscopy Describe	TRBC x106cells/cu mm
86	130/90	22	hemic murmur, splenomegaly		splenomegaly		normal	2.2
70	130/80	20	moderate splenomegaly		hepatosplenomegaly		normal	1.83
92	150/80	14	sive splenomegaly		splenomegaly		normal	2.12
76	110/60	14	hemic murmur, B/L crepts, splenomegaly		old PT, Splenomegaly, increased renal cortical echoes		pale upper GI mucosa	1.8

93	140/70	22	NAD	mild splenomegaly	normal	1.92
88	120/70	22	NAD	cardiomegaly	normal	2.63
66	130/100	26	NAD	fatty liver	normal	2.39
82	110/80	16	ca breast	NAD	normal	3.11
71	140/70	19	ild splenomegaly	fatty liver mild splenomegaly	congested GI	1.19
80	150/70	14	ystolic murmur	cardiomegaly B/L minimal PE	pale UGI muc	2.23
76	110/70	22	mild hepatomegaly, B/L crepts	NAD	pale upperGIr	3.1
84	130/70	20	NAD	cardiomegaly	normal	3
68	130/90	24	hemic murmur	mild cardiomegaly	normal	3.3
78	160/100	23	der hepatomegaly	B/L medical renal disease type III RPD changes	normal	2.68

68	140/90	20	hemic murmur	old PT cardiomegaly	pale UGI muc	1.56
76	140/70	16	b/l crepts	cardiomegaly mild pleural effusion minimal ascitis	normal	1.01
74	120/80	16	NAD	cerebral atrophy	normal	2.2
70	11o /70	23	hemic murmur, thyromegaly	B/L MRD type I CKD	normal	2.1
100	110/70	20	ld hepatomegaly	consolidation lung early type I MRD mild hepatomegaly	pale UGI muc	1.27
86	140/80	20	NAD	cardiomegaly splenomegaly	normal	1.82
94	160/90	22	hemic murmur	cardiomegaly mildsplenomegaly,B/L moderate PE	ulcer, incompetent GEJ	1.36

78	120/70	18	NAD	NAD	pale UGImuc	2.5
96	130/80	15	hemic murmur	cardiomegaly moderate splenomegaly	pale upper GI	1.95
84	130/80	16	hemic murmur	NAD	pale upperGIr	3
94	140/80	16	NAD	normal	growth body fundus of stomach up to LES	2.47

TLC x103cells/cu mm	PLC x105cells/cu mm	HB gms/dl	HCT	MCV	MCH	MCHC	RDW-SD	Retic C
145	0.28	6.9	18.9	85.9	31.5	36.5		0.5
1.53	3.24	6.7	22.1	106	23.2	26.9		0.5
119	1.74	8.9	20.9	63.7	21.3	27.4		0.5
125	3.19	7.7	21.3	83.3	26.1	31.3		1

2.9	71000	4.8	16	83.3	24.5	29.4	1
5.9	2.94	9.1	29.6	100.4	22.6	28.3	0.5
4.3	2.69	6.6	22.1	94	23.8	27.4	0.5
4.8	2.45	7.6	22.3	84.7	24.5	30.2	0.5
11.4	1.73	8.1	24.9	116.9	32.7	31.3	1.5
6.4	2.11	8.3	20.6	98.4	22.1	28.4	0.5
6.2	1.5	6.1	18	102.4	23.2	26.8	0.5
4.5	4.22	7	24	66	18.7	22.6	<0.5
12	3.3	9.1	36	89.4	23.6	30.1	<0.5
4.5	2.31	8.2	24.7	99.6	30.6	30.8	0.5

3.2	0.9	6.8	19.6	92.5	18.7	26.6	0.5
5.8	2.92	6.8	16.6	65.3	15.8	24.2	0.5
3	0.3	6.5	20.1	91	29.4	32.3	1
3.9	2.12	5.6	24	76.4	21.6	28.8	1
4	10000	4.7	13.9	90.1	32.2	35.8	0.5
2.5	30000	4.9	16.7	129.3	41.5	32.1	1
11.9	3.5	5.4	18	132.4	26.7	30	1
6.5	3.26	8.3		78.5			0.5
5.3	2.84	10.8		90.4			0.5
7.8	2.78	7.5		83.2			1.4
7.2	1.79	10.4		88.3			1
4.7	3.16	9.3		87.4			0.5

6.9	2.5	5.6	16	89.3	23.6	27.9	0.3
7.3	1.42	5.1	13.8	70.8	16.4	23.2	2
7.4	2.53	8.7		82.6			1.2
10.5	1.8	6.2	26	74	22	22	1
6.4	5.63	6.9	19.6	79	23.5	29.6	1

ESR 1Hr	BSR	BUN	SCR	SBRT	SGOT	SGPT	ALP	SPROT
10 50	92	65	3	0.8	28	26	84	6.2
60	73	38	1.2	0.5	62	66	110	4.9
98 130	70	30	1.1	0.7	20	22	102	4.6
50 100	68	20	0.8	0.9	18	23	96	7.3

45, 85	65	41	1.2	3.8	48	24	65	6.7
50	94	23	0.9	0.6	34	36	102	5.4
50 130	146	38	1.1	0.6	18	29	107	5.3
110	102	36	0.9	1	26	28	88	6.2
110 150	106	29	0.8	0.9	32	21	96	6.2
100	75	41	1.1	1.2	37	44	107	5.6
52	35 3	28	1.3	0.6	18	22	86	5.8
5, 20	75	16	1.3	0.5	18	21	88	5.9
55 110	86	40	0.8	0.9	21	32	97	6.2
20 130	157	150	5.4	1.1	46	39	114	4.8

40	92	19	1	1.1	16	21	76	6.2
32 60	98	63	1.2	2.7	105	123	197	5.8
100 130	65	50	1	1	38	27	79	6.3
50, 105	96	129	2.2	1.2	36	26	206	5.3
100 150	125	22	1	0.6	31	31	85	7.9
28/50	128	30	1.1	2.1	21	28	90	4.6
100 130	114	44	1.2	2.4	19	22	85	5.3

30 50	106	22	1.1	0.6	36	29	99	5.3
30 60	146	21	0.8	1.1	41	44	118	4.8
55 120	148	24	0.8	1	40	36	104	5.7
50 100	96	25	0.8	0.4	19	41	98	6.6

ALB	T3	T4	TSH	PSS-RBC Describe RBC	PSS-RBC Impression	PSS-RBC 1- Normocytic 2-Microcytic 3-Macrocytic 4-Dimorphic 5-Others	PSS-WBC Describe	PSS-WBC Impression
3.3		1.7	88	0.8 number decreased microcytic hypochromic RBCs	MCHC anemia	2	increased myelolasts	leukemia
2.2		0.9	78	0.7 number decreased macrocytic RBCs	macrocytic anemia	3	number increased mature neutrophils, band forms myelocytes occasssional blasts	CML - chronic phase
2.7		1.6	82	0.9 number decreased microcytic hypochromic RBCs	MCHC anemia	2	increased in neutrophils band forms occassional blasts	CML - chronic phase
4.3		1	92	3 number decreased microcytic hypochromic RBCs	MCHC anemia	2	increase in mature neutrophil, band forms, myelocyt basophils	CML- accelerated phase

3.2	1.2	89	2.3	number decreased microcytic n macrocytic RBCs	dimorphic anemia	4	normal in number n morphology	normal
3	1.2	66	0.4	number decreased microcytic n macrocytic RBCs	dimorphic anemia	4	normal number n morphology	normal
2.2	1.1	86	0.7	number decreased normocytic n macrocytic RBCs	macrocytic anemia	3	normal number n morphology	Normal
2.9	0.7	74	1.9	number decreased normocytic BBCs	normocytic anemia	1	normal number n morphology	Normal
3.2	68	4.5	2.1	number decreased normocytic n macrocytic RBCs	macrocytic anemia	3	increase in neutrophils	reactive neutrophilia
3.2	0.5	79	0.4	number decreased microcytic n macrocytic RBCs	dimorphic anemia	4	normal number n morphology	Normal
3.1	1.1	83	0.7	number decreased macrocytic RBCs	macrocytic anemia	3	normal number n morphology	Normal
2.7	1.3	78	0.3	number decreased macrocytic RBCs	macrocytic anemia	3	normal in number n morphology	Normal
2.9	1.9	52	3.5	number decreased microcytic n macrocytic RBCs	dimorphic anemia	4	number increased shift to left	reactive neutrophilia
2.8	0.7	74	1.9	number decreased microcytic and macrocycytic RBCs	dimorphic anemia	4	increase in neutrophils	RN

3.1	1.2	65	0.7	number decreased macrocytic RBCs	macrocytic anemia	3	number decreased	leucopenia
3	1.5	61	0.5	number decreased microcytic n normocytic	dimorphic anemia	4	normal in number n morphology	normal
3.2	2	92	3.3	number decreased microcytic n normocytic RBCs	dimorphic anemia	4	number decreased	leucopenia
2.6	1.3	86	0.6	number decreased microcytic and macrocytic RBCs	dimorphic anemia	4	normal in number n morphology	normal
4.4	1.2	89	1	number decreased microcytic hypochromic RBCs	MCHC anemia	2	number decreased	leucopenia
2.5	1.3	103	1.1	number decreased microcytic hypochromic RBCs	MCHC anemia	2	number decreased	leucopenia
2.2	1.7	89	1	number decreased microcytic hypochromic RBCs	MCHC anemia	2	increase in neutrophils toxic vacuoles	reactive neutrophia
					MCHC anemia	2		Normal
					normocytic anemia	1		Normal
					MCHC anemia	2		RN
					MCHC anemia	2	n	RN
					normocytic anemia	1		RN

2.8	0.9	78	0.9	number decreased normocytic n microcytic RBCs anisopoikilocytosis	dimorphic anemia	4	increase in neutrophils	RN
2.6	1.8	88	0.6	number decreased microcytic hypochromic RBCs	MCHC anemia dimorphic anemia	2 4	normal number n morphology	normal RN
3.1	1.6	82	1.2	number decreased microcytic hypochromic RBCs	MCHC anemia	2	normal in number n morphology	RN
3.2	1.8	76	0.3	number decreased microcytic hypochromic RBCs	MCHC anemia	2	normal in number n morphology	normal

PSS-WBC 0-Normal 1-Reactive 2-Suspicious 3-Malignancy 4-Inconclusive	PSS-PL Describe	PSS-PL Impression	PSS-PL 0-Normal 1-High 2-Low 3-Giant Pl	Marrow Cellularity 0-Normal 1-Hypo 2-Hyper	Marrow Erythroid Describe	Marrow Myeloid Describe	Marrow Megk Describe	Marrow Others Describe
3	number reduced	thrombocytopenia	2	2	number decreased micronormoblastic maturation	increased in number myeloblasts >90%	decreased	
3	number increased normal in morphology	reactive thrombocytosis	1	2	number decreased micro n macronormoblastic maturation	increase in myeloblasts promyelocytes myelocytes band forms mature neutrophils	normal in number n morphology	
3	number increased normal morphology	thrombocytosis	1	2	number decreased micronormoblastic maturation	increase in myeloblasts promyelocytes metamyelocytes	normal number n morphology	
3	number increased normal morphology	thrombocytosis	1	2	number decreased micronormoblastic maturation	increase in myeloblasts promyelocytes metamyelocytes basophils	number increased normal morphology	

0	normal in number n morphology	normal	0	0	number increased megaloblastic maturation	megaloblastic maturation	normal number n morphology
0	normal number n morphology	normal	0	0	number normal megaloblastic maturation	number normal giant metamyelocytes band forms	normal number n morphology
0	normal number n morphology	normal	0	0	number increased megaloblastic maturation	normal number n maturation	normal number n morphology
0	normal number n morphology	normal	0	0	number increased megaloblastic maturation	normal number n maturation	normal number n morphology
1	normal number n morphology	normal	0	0	number increased megaloblastic maturation	normal number normal maturation	normal number n morphology
0	normal number n morphology	normal	0	2	number increased megaloblastic maturation	number increased megaloblastic maturation	normal number n morphology
0	normal number n morphology	normal	0	2	number increased megaloblastic maturation	number increased giant metamyelocytes	normal number n morphology
0	normal in number n morphology	normal	0	2	number increased megaloblastic maturation	number increased myelocyte metamyelocyte giant band forms	normal in number n morphology
1	normal number n morphology	normal	0	2	number increased megaloblastic maturation	increased giant metamyelocytes band forms	normal number n morphology
1	normal number and morphology	normal	0	2	number increased megaloblastic maturation	increase in metamyelocytes giant band forms	normal in number n morphology

2	number decreased normal morphology	thrombocytopenia	2	2	number increased megaloblastic maturation	number increased megaloblastic maturation	normal number n morphology	
0	normal in number n morphology	normal	0	1	micro normoblastic maturation dyserythropoiesis	normal maturation	dysmorphic megks	plasmacytosis
2	number decreased normal morphology	thrombocytopenia	2	1	number decreased dyserythropoiesis	number decreased dysmyelopoiesis	normal number dysmorphic megks	
0	normal in number n morphology	normal	0	2	number increased megaloblastic maturation	number increased dysmyelopoiesis	normal in number n morphology	
2	number decreased	thrombocytopenia	2	2	number decreased dyserythropoiesis	number increased dysmyelopoiesis	reduced number dysmorphic forms	
2	number reduced normal morphology	thrombocytopenia	2	2	number decreased micro n macronormoblastic maturation number decreased dividing cells dyserythropoiesis	number increased myeloblasts promyelocytes mature forms	variable	
1	number reduced normal morphology	thrombocytopenia	2	2	dividing cells dyserythropoiesis	dysmyelopoiesis	normal morphology	
0	n	normal	0	0				
0		normal	0	0				
1		normal	0	0				
1		normal	0	0				
1	n	normal	0	0				

1	number increased normal in morphology	reactive thrombocytosis	1	0	number increased micro n macro normoblastic maturation	incresed in number normal maturation	normal in number n morphology	
0	normal number n morphology	normal	0	2	number increased, micro and macro normoblasti maturation	normal number n morphology	normal number n morphology	plasmacytosis
1		normal	0	2				
1	normal in number n morphology	normal	0	2	number increased micro n macro normoblasti maturation	normal in number n morphology	normal in number n morphology	
0	number increased normal morphology	reactive thrombocytosis	1	2	number increased micronormoblastic maturation	normal number n morphology	normal number n morphology	

Marrow Impression Describe	diagnosis
AML-M1	AML
CML - chronic phase	CML
CML chronic phase	CML acc.
CML - accelerated phase	CML

megaloblastic anemia	meg ane
megaloblastic anemia	meg ane
reactive marrow with megaloblastic maturation	meg ane
megaloblastic anemia	meg ane
erythroid hyperplasia withmegaloblastic maturation consistent with megaloblastic anemia	meg ane
megaloblastic anemia	meg ane
megaloblastic anemia	meg ane
megaloblastic anemia	meg ane
megaloblastic anemia	meg ane
megaloblastic anemia	meg ane

megaloblastic anemia meg ane

MDS with plasmacytosis - RCMD

MDS RCMD megaloblastic marrow with dysmyelopoiesis s/o MDS-RA

MDS trilineage dysplasia - RAEB

MDS RCMD

MDS RCMD combined deficiency combined
combined deficiency combined
combined deficiency combined
combined deficiency combined
combined deficiency combined

reactive marrow with micro n macronormo blastic maturation s/o combined deficiency	combined
REH with micro n macro normoblasti maturation and plasmacytosi s	combined
combined deficiency	combined
REH with micro n macro normoblastic maturation s/o combinrd deficiency	combined
REHs/o IDA	combinedIDA

6.15779	kosalai	65	F	fatigue	1,2,3	7	.	0
14. 45911	madathi	65	F	hemoptysis, fever	15	7	2	3
8.4471	santhivinayag	65	M	ulcer leg	..	7	2	1, 3
36. 42317	ganapathy	66	M	loss of apetite fatigue	15	7	2	2
4.4798	esther	62	F	abdominal pain dyspnoea	1,2, 15	7	.	0
31. 15364	patrakali	66	F	exertional dyspnoea	. .	7	2	3
17. 42094	subbiah	60	M	dyspnoea decreased urine output giddiness	2, 15	7	2	1
23. 45982	srinivasan	74	M	swaying while walking	2	7	2	2,3
19. 49305	arumugam	76	M	facial puffiness pedal edema	1,5	7	2	. .
29. 6858	gandhi	85	M	hematemesis	. .		1	. .

					2			
20.	46728	esakki	72	M	weakness limb		7	2	1, 2, 3
					3,8,10,15				
15.	43507	gurusamy	70	M	dyspnoea		7	2	3
					1				
48.	76231	subbulakshmi	61	F	vomiting fever		1, 2	2	0
	10.2039	mariyal	63	F	giddiness exertional dyspnoea	1,2,, 6	3, 7	2	0
						4,11			
27.	33513	vellathai	65	F	abdominal pain icterus		7	2	0
						15			
50.	22754	saroja	68	F	pedal edema facial puffiness		7	2	3
22.	40029	veeramariamn	70	F	fever abdominal pain	2,5,	7	2	. .
	1.5654	saraswathy	77	F	dyspnoea, pedal edema decreased urine output	1	7	. .	0
						15			
43.	3904	velayutham	65	M	chestpain giddiness		7	. .	2

7. 12487	subramanian	65	M	dyspnoea chestpain	2, 15	7	. .	2,3
46. 4954	krishnan	69	M	giddiness pedal edema	10, 15	7	2	3
13. 43705	madasamy	70	M	pedal edema dyspnoea	2, 3	7	2	. .
33. 47768	muthukrishna	72	M	giddiness pedal edema	2,3,6	7	2	.?
66		63	F	chest pain, exertional dyspnoea				
2.6784	saraswathy	65	F	mass P/V urinary disturbances	1	7	2	3
3.1364	seetha	65	F	dyspnoea on exertion	5	7	2	3
60		73	F	pedal eema chest pain fatigue	15			
63		60	M	exertional dyspnoea	1			
56		61	M	giddiness	. .			

18.	44867	mariappan	62	M	pedal edema facial puffiness	2	7	2	3
	67	selvaraj	62	M	easy fatiguability loss of apetite	’ ’			
	57	sudalaimuthu	73	M	giddiness	4			
	62		81	F	loss of apetite vomiting fatigue	..			
5.	1400	pathmanaban	61	M	pedal edema abdominal pain	2	7	1	0
24.	47881	paramasivan	62	M	exertional dyspnoea pedal edema	1,2	7	2	2
16.	61113	shanmugasun	62	M	cough palpitation pedal edema	5 , 10	7	2	0
25.	48220	murugan	63	M	fever	15	7	2	2
28.	14794	sundar raj	69	M	giddiness	2, 10	7	2	1
41.	31693	kannimalar	70	M	giddiness	2, 3, 10	7	1	3
21.	50914	esakkkiamma	66	F	mass abdomen	5, 8	7	2	3

49.	50267	ponnammal	66	F	exertional dyspnoea palpitation	15	7	2	3
	54		66	F	fatigue	3			
	58	velu	67	F	giddiness loss of apetite fever	, ,			
	68		71	F	fatigue vomiti	. .			
	65		74	F	giddiness, chestpain palpitation				
40.	37152	lakshmi	60	F	abdominal pain vomiting	8	2	2	1
37.	4632	rajeshwari	63	F	easy fatiguability loss of apetite	12, 14	1,2, 7	2	3
	9.9578	bagavathy	66	F	fatigue	5, 8	7	. .	3
11.	60969	lakshmi	66	F	fatigue diarrhc	14	7	2	3
39.	19917	ramani	67	F	loss of apetite	12	2	2	1

					8, 12, 14			
45. 3398	sudalaimadan	61	M	abdominal pain malena		2, 7	2	2
					8			
42. 14794	soundarajan	62	M	exertional dyspnoea palpitation		7	2	2
					1,2,3			
34. 49335	manoharan	65	M	fever, abdominal pain		7	2	3
					2,.15			
26. 32431	poothathan	65	M	abdominal pain dyspnoea		7	, ,	. .
					2,4			
47. 3490	jesumichel	65	M	pedal edema loss of weight		7	2	2,3
					…			
	55	60	F	giddiness			2	,
					1,2,4,6			
32. 50536	kanchana	61	F	abdominal pain icterus		3, 7	1	0
					1			
	64	61	F	fatigue chest pain giddiness				
					2, 5, 6			
44. 3422	shanmugathar	65	F	exertional dyspnoea pedal edema		7	1	0
				loss of apetite vomiting	..			
	62	81	F	fatigue				

3	0	2	0	0	86	130/90	22	hemic murmur, splenomegaly	splenomegaly
1	0	2	0	1	76	110/60	14	hemic murmur, B/L crepts, splenomegaly	old PT, Splenomegaly, increased renal cortical echoes
2	0	2	0	1	92	150/80	14	moderate splenomegaly	splenomegaly
1	1	2	0	0	70	130/80	20	moderate splenomegaly	hepatosplenomegaly cardiomegaly mild
3	0	1	0	0	86	140/80	20	NAD	splenomegaly consolidation lung early type I MRD
3	0	0	1	1	100	110/70	20	mild hepatomegaly	mild hepatomegaly
3	0	0	0	1	94	160/90	22	hemic murmur	cardiomegaly mildsplenomegaly,B/L moderate PE
3	0	0	0	1	74	120/80	16	NAD	cerebral atrophy cardiomegaly mild pleural effusion minimal ascitis
1	0	0	0	1	76	140/70	16	b/l crepts	
3	0	0	0	0	70	11o /70	23	hemic murmur, thyromegaly	B/L MRD type I CKD

2	0	0	0	1	80	150/80	18	spastic quadriparesis	non compressive myelopathy
3	0	0	0	1	85	120/ 70	18	systolic murmur B/L crepts	old PT B/L medical renal disease type I RPD changes
2	0	0	0	0	82	110/80	16	ca breast on treatment	NAD
1	0	0	0	1	66	130/100	26	NAD	fatty liver
2	1	1	0	0	71	140/70	19	mild splenomegaly	fatty liver mild splenomegaly
1	0	0	0	1	80	150/70	14	systolic murmur	cardiomegaly B/L minimal PE
1	0	0	0	0	68	130/90	24	hemic murmur	mild cardiomegaly
3	0	0	1	1	76	110/70	22	mild hepatomegaly, B/L crepts	NAD
1	0	0	0	1	88	120/70	22	NAD	cardiomegaly

3	0	0	0	0	84	130/70	20	NAD	cardiomegaly
3	0	0	0	1	68	140/90	20	hemic murmur	old PT cardiomegaly
2	1	0	1	1	78	160/100	23	tender hepatomegaly	B/L medical renal disease type III RPD changes
1	1	0	0	1	93	140/70	22	NAD	mild splenomegaly
3	0	0	0	0	78	120/70	18	NAD	NAD
1	0	0	0	0	84	130/80	16	hemic murmur	NAD

1	0	0	1	1	96	130/80	15	hemic murmur	cardiomegaly moderate splenomegaly
3	0	0	0	1	96	140/90	26	NAD	mild splenomegaly
2	0	1	1	1	73	170/100	20	ESM, hepatosplenomegaly	mild cardiomegaly
1	0	0	2	1	84	110/80	14	epigatric tenderness hepatomegaly	old PT
1	0	0	0	0	104	140/80	26	NAD	NAD
1	0	0	0	1	110	130/80	22	systolic murmur	NAD
2	1	0	0	0	63	120/80	16	NAD	old PT B/L small kidneys
1	0	3	1	0	88	100/70	18	liver just palpable massivesplenomegaly	cardiomegaly splenomegaly dilated portal vein

3	0	0	0	1	78	140/80	23	b/l crepts	pneumonitic changes mild cardiomegaly
1	0	0	0	0	74	130/90	22	epigastric mass	antral growth simple hepatic cyst
1	0	0	0	0	84	120/ 80	18	ca breast	NAD
2	1	2	1	1	72	120/60	15	splenomegaly, tender hepatomegaly	splenomegaly, renal calculi
1	1	0	2	1	90	120/80	16	hepatomegaly	hepatomegaly
1	0	0	0	0	68	14/ 80	18	/c/o ca alveolu	NAD

1	1	0	0	0	62	140/60	19	/c/o ca stomacl	NAD
3	0	0	0	0	78	150/60	21	hemic murmur, haemorrhoids	NAD
2	0	2	1	1	90	120/80	16	moderate splenomegaly tender hepatomegaly	hepatosplenomegaly
3	0	1	0	1	66	150/90	22	splenomegaly	splenomegaly cholelithiasis renal calculi
1	0	1	1	1	92	120/80	22	jaundice hepatomegaly	hepatosplenomegaly
2									
2	0	1	1	1	94	130/80	16	jaundice hepatomegaly	splenomegaly calculous cholecystitis
3	1	1	0	1	76	110/80	16	ld splenomega	splenomegaly cardiomegaly

not done	2.2	145	0.28	6.9	18.9	85.9	31.5	36.5
not done	1.8	125	3.19	7.7	21.3	83.3	26.1	31.3
not done	2.12	90,000	1.74	8.9	20.9	63.7	21.3	27.4
not done	1.83	1.53	3.24	6.7	22.1	106	23.2	26.9
not done	1.82	2.5	30000	4.9	16.7	129.3	41.5	32.1
pale UGI muc	1.27	4	10000	4.7	13.9	90.1	32.2	35.8
ulcer, incompetent GEJ	1.36	11.9	3.5	5.4	18	132.4	26.7	30
not done	2.2	3	0.3	6.5	20.1	91	29.4	32.3
normal	1.01	5.8	2.92	6.8	16.6	65.3	15.8	24.2
normal	2.1	3.9	2.12	5.6	24	76.4	21.6	28.8

not done	1.63	1200	1.27	7.7	25.6	104	31.3	30.1
pale upper GI	2.5	3.2	0.4	6.8	22.4	128	27	32
not done	3.11	4.8	2.45	7.6	22.3	84.7	24.5	30.2
not done	2.39	4.3	2.69	6.6	22.1	94	23.8	27.4
congested GI	1.19	11.4	1.73	8.1	24.9	116.9	32.7	31.3
pale UGI muc	2.23	6.4	2.11	8.3	20.6	98.4	22.1	28.4
not done	3.3	12	3.3	9.1	36	89.4	23.6	30.1
pale upperGIr	3.1	6.2	1.5	6.1	18	102.4	23.2	26.8
not done	2.63	5.9	2.94	9.1	29.6	100.4	22.6	28.3

normal	3	4.5	4.22	7	24	66	18.7	22.6
not done	1.56	3.2	0.9	6.8	19.6	92.5	18.7	26.6
normal	2.68	4.5	2.31	8.2	24.7	99.6	30.6	30.8
not done	1.92	2.9	71000	4.8	16	83.3	24.5	29.4
	1.88	6.5	3.26	8.3	36.8	86.5	22.6	28.7
not done	2.5	6.9	2.5	5.6	16	89.3	23.6	27.9
not done	3	10.5	1.8	6.2	26	74	22	22
not done		5.3	2.84	10.8		90.4		
not done		7.8	2.78	7.5		83.2		
not done		4.7	3.16	9.3		87.4		

not done	1.95	7.3	1.42	5.1	13.8	70.8	16.4	23.2
		7.2	1.79	10.4		88.3		
		7.4	2.53	8.7		82.6		
not done		6.3	1.85	6.8		79		
gastric mucosal prolapse	3.47	5	2.98	5.3	22.3	64.3	15.3	23.8
congested GI	2.19	4.3	1.35	6.3	20.3	92.7	28.8	31.1
	2.28	7.8	2.95	5.9	14.4	63.2	14.9	23.6
normal	3.2	5.8	90	10.4	33.7	97.4	32.9	33.8
pale UGI muc	2.34	5.3	2.05	6.4	28.1	73.4	24.5	26.2
pale UGI muc	2.09	5.4	2.31	7.1	26.4	65.5	16.7	25.1
esophageal varices severe PHT/ gastropathy	2.22	4.2	0.41	6.3	18.4	93.6	18.4	22.3

not done	1.87	9.2	1.48	5.7	19.4	76.9	21.5	27.3
not done		5.6	2.29	8.2		69.6		
not done		2.9	1.13	5.7		62.9		
not done		3.8	1.06	11.2		77.6		
		4.1	1.52	7.7		86.6		
proliferative growth pylorus	3.1	6000	3.8	5.7	19.4	73.5	22.7	28.5
not done	2.2	6.5	2.98	10.3	32.3	88.7	25.6	32.1
	2.7	11	0.19	6.4	19.3	72.5	21.3	31.1
	1.96	10.6	3.55	5.8	20.1	66.4	21.2	29.6
not done	2.88	6.5	3.32	10.2	33.1	66.6	26.2	29.7

not done	2.26	5.2	2.13	6.2	24.8	68.7	23.2	25.7
not done	2.99	6.3	4.32	8.9	30.1	78.9	21.2	32.2
not done	2.67	7.3	1.56	7.5	23	86.4	28.1	34.6
pale UGI mucosa	2.26	34.5	1.1	6.5	27.6	72.8	17.8	24.5
	2.99	7.3	2.65	8.3	20.2	56.5	19.3	22.4
		6.1	3.41	6.9		74.8		
	3.2	9.9	4.1	8.7	30.6	72.9	19.6	26.9
not done		5.3	2.73	6.3		59.7		
normal	1.78	4.7	3.96	7.4	23.5	77.2	19.3	27.4
not done		6.3	1.85	6.8		81.7		

. .	.	92	65	3	. .	. .	..
. .	60	73	38	1.2	0.5	62	66
. .	. .	68	20	0.8	.	.	.
. .	98 130	70	30	1.1	0.7	20	22
0.5	..	113	43	0.6	..	.	.
0.5							
1.4							
0.5		74					
1							
1.2							

1.5	110 150	106	29	0.8	0.9	32	21
1.1	10 22	137	27	0.7	2.4	63	54
1.2							
0.6							
0.5							
1	28/50	128	30	1.1	2.1	21	28
1	100 130	114	44	1.2	2.4	19	22
1	100 130	65	50	1	1	38	27
0.5	100 150	125	22	1	0.6	31	31

0.5	32 60	98	63	1.2	2.7	105	123
1.2	150 165	138	54	2	0.7	22	28
0.5	10 40	135	23	0.8	1.1	62	28
0.5	110	102	36	0.9	1	26	28
0.5	. .	146	38	1.1	0.6	18	29
0.5	100	75	41	1.1	1.2	37	44
<0.5	55 110	86	40	0.8	0.9	21	32
0.5	52	35 3	28	1.3	0.6	18	22
0.5	50	94	23	0.9	0.6	34	36
<0.5	5, 20	75	16	1.3	0.5	18	21

0.5	40	92	19	1	1.1	16	21
0.5	20 130	157	150	5.4	1.1	46	39
1	45, 85	65	41	1.2	3.8	48	24
1	50, 105	96	129	2.2	1.2	36	26
0.5							
0.6	40 85	89	32	0.9	0.8	34	28
1.5	50, 100	74	24	0.8	1	42	36
1	100 150	104	29	0.9	0.8	41	36
< 0.5	55 100	137	44	1	1.6	25	41
1.3	100 132	132	95	4.3	0.6	40	20
1	48	66	22	0.8	0.7	34	42

1							
0.3							
1	15 30	60	44	1.2	0.8	17	24
0.5							
0.5	42	60	48	1.3	0.7	28	33
0.3	30 50	106	22	1.1	0.6	36	29
3	26 80	76	16	0.6	1.2	34	22
0.5	55	64	21	0.6	0.9	18	26
0.5	50 105	117	76	1.4	0.6	26	26
1	80 120	78	36	0.9	1.1	.	.

1	130	112	35	1.1	1.2	26	41
0.5	30 50	112	42	0.9	1.1	20	24
1	55 120	148	24	0.8	1	40	36
2	30 60	146	21	0.8	1.1	41	44
1.3	10 22	115	34	1.5	0.4	34	42
0.5	55 100	146	20	0.8	0.9	40	36
0.5	50	76	28	0.6	0.9	18	21
1.2	60	97	20	0.9	0.7	19	22
0.5	50 100	110	28	0.8	.	.	.
1.2							

. .	. .	. .		0	0	0	number decreased microcytic hypochromic RBCs	MCHC anemia	2
110	4.9	2.2	.	.	.		number decreased macrocytic RBCs	macrocytic anemia	3
.	.	. .		0	0	0	number decreased microcytic hypochromic RBCs	MCHC anemia NRBC	2
102	4.6	2.7	.	.	.		number decreased microcytic hypochromic RBCs	MCHC anemia	2
.	.	.	.	.	.			MCHC anemia	2
								normocytic anemia	1
								MCHC anemia	2
								normocytic anemia	1
								MCHC anemia mild anisocytosis	2
								dimorphic anemia	4

96	6.2	3.2		68	4.5	2.1	number decreased normocytic n macrocytic RBCs	macrocytic anemia	3
109	4.8	3.1		0.5	78	0.9	number decreased schistocytes	hemolytic blood picture MCHC	2
						0.7		dimorphic anemia	4
						1.3		MCHC anemia	2
						0.4		normocytic anemia	1
90	4.6	2.5	.	.	.		number decreased microcytic hypochromic RBCs	MCHC anemia	2
85	5.3	2.2		1.7	89	1	number decreased microcytic hypochromic RBCs	MCHC anemia	2
79	6.3	3.2		2	92	3.3	number decreased microcytic n normocytic RBCs	dimorphic anemia	4
85	7.9	4.4	.	.		1	number decreased microcytic hypochromic RBCs	MCHC anemia	2

197	5.8	3		1.5	61	0.5	number decreased microcytic n normocytic	dimorphic anemia	4
101	5.6	3		1.3		0.3	number decreased macrocytic n normocytic RBCs	dimorphic anemia	4
84	6	2.7		1.6	81	1	number decreased microcytic n macrocytic RBCs	macrocytic anemia	3
88	6.2	2.9		0.7	74	1.9	number decreased normocytic BBCs	normocytic anemia NRBC	1
107	5.3	2.2		1.1	86	0.7	number decreased normocytic n macrocytic RBCs	macrocytic anemia	3
107	5.6	3.2	.	.	.		number decreased microcytic n macrocytic RBCs	dimorphic anemia	4
97	6.2	2.9	. .	. .		3.5	number decreased microcytic n macrocytic RBCs	dimorphic anemia	4
86	5.8	3.1		1.1	83	0.7	number decreased macrocytic RBCs	macrocytic anemia	3
102	5.4	3		1.2	66	0.4	number decreased microcytic n macrocytic RBCs	dimorphic anemia	4
88	5.9	2.7		1.3	78	0.3	number decreased macrocytic RBCs	macrocytic anemia	3

76	6.2	3.1	.	.		0.7	number decreased macrocytic RBCs	macrocytic anemia	3
114	4.8	2.8	.	.	.		number decreased microcytic and macrocycytic RBCs	dimorphic anemia	4
65	6.7	3.2		1.2	89	2.3	number decreased microcytic n macrocytic RBCs	dimorphic anemia	4
206	5.3	2.6		1.3	86	0.6	number decreased microcytic and macrocytic RBCs	dimorphic anemia	4
						0.8		MCHC anemia	2
116	6.1	2.9	.	.	.		number decreased microcytic hypochromic RBCs	MCHC anemia anisopoikilo cytosis	2
151	8.6	4.6	.	.	.		number decreased normoctic normochrom ic RBCs	normocytic anemia	1
112	5.2	2.9		0.5	74	0.8	number decreased microcytic hypochromic RBCs	MCHC anemia	2
109	7	4		1.4	70	0.5	number decreased microcytic and macrocytic RBCs	MCHC anemia	2
104	7.1	3	.	.		1.1	number decreased microcytic hypochromic RBCs	MCHC anemia	2
92	5.8	2.7		1.1	83	0.7	number decreased microcytic hypochromic RBCs	MCHC anemia	2

						0.6		MCHC anemia	2
						1.7		MCHC anemia	2
89	5.4	2.3	.	.	.		number decreased normocytic normochromic RBCs occassional macrocyte	normocytic anemia	1
						1.9		normocytic anemia	1
89	5.6	2.8	.	.	.		number decreased microcytic n macrocytic RBCs	MCHC anemia	2
99	5.3	2.8	.	.	.		number decreased normocytic n microcytic RBCs anisopoikilocytosis	dimorphic anemia	4
107	6.1	3		1.3	91	0.4	number decreased microcytic hypochromic RBCs	dimorphic anemia	4
100	4.9	2.7		0.9	91	0.3	number decreased microcytic hypochromic RBCs	MCHC anemia	2
145	5.9	2.5					number decreased microcytic hypochromic RBCs	MCHC anemia	2
.	.	.	.	.	.		number decreased normocytic normochromic RBCs	normocytic anemia	1

101	6.3	3.2	1.5	61	0.5	number decreased microcytic hypochromic RBCs	MCHC anemia	2
88	5.3	3.2				number decreased microcyti hypochromic RBCs	MCHC anemia	2
104	5.7	3.1	1.6	82	1.2	number decreased microcytic hypochromic RBCs	MCHC anemia anisopoikilo cytosis	2
118	4.8	2.6	1.8	88	0.6	number decreased microcytic hypochromic RBCs	MCHC anemia	2
98	6.1	3.1	1.2	83	0.5	number decreased microcytic hypochromic RBCs	MCHC anemia	2
106	5.2	3.2	0.8	88	1.2	number decreased microcytic hypochromic RBCs	MCHC anemia	2
87	5.4	2.5	1.3	91	0.4	number decreased microcytic hypochromic RBCs	MCHC anemia	2
94	6.1	3.2	1.2	76	0.6	number decreased microcytic hypochromic RBCs	MCHC anemia	2
.	.	.	1.2	65	0.7	number decreased microcytic hypochromic RBCs	MCHC anemia	2
					0.7		dimorphic anemia	4

increased myelolasts	acute leukemia	3	number reduced	thrombocytopenia	2	2	number decreased micronormoblastic maturation	increased in number myeloblasts >90%
number increased mature neutrophils, band forms myelocytes occasssional blasts	CML - acc.phase	3	number increased normal in morphology	reactive thrombocytosis	1	2	number decreased micro n macronormoblastic maturation	increase in myeloblasts promyelocytes myelocytes band forms mature neutrophils
increase in mature neutrophil, band forms, myelocyt basophils	CML-accelerated phase	3	number increased normal morphology	thrombocytosis	1	2	number decreased micronormoblastic maturation	increase in myeloblasts promyelocytes metamyelocytes basophils
increased in neutrophils band forms occassional blasts	CML - chronic phase	3	number increased normal morphology	thrombocytosis	1	2	number decreased micronormoblastic maturation	increase in myeloblasts promyelocytes metamyelocytes
	Normal	0	n	normal	0	0		
	Normal	0		normal	0	0		
	RN	1		normal	0	0		
	RN	1	n	normal	0	0		
n	RN	1		normal	0	0		
	RN	1		normal	0	2		

increase in neutrophils	reactive neutrophilia	1	normal number n morphology	normal	0	0	number increased megaloblastic maturation	normal number normal maturation
normal number n morphology	RN	1	number increased	reactive thrombocytosis	1	0	number increased micronormoblastic maturation	normal number n maturation
	normal	0		normal	0	2		
	normal	0		reactive thron	1	0		
	normal	0		normal	0	0		
number decreased	leucopenia	2	number reduced normal morphology	thrombocytopenia	2	2	number decreased micro n macronormoblastic maturation dividing cells dyserythropoiesis	number increased myeloblasts promyelocytes mature forms
increase in neutrophils toxic vacuoles	reactive neutrophia	1	number reduced normal morphology	thrombocytopenia	2	2	dividing cells dyserythropoiesis	dysmyelopoisis
number decreased	leucopenia	2	number decreased normal morphology	thrombocytopenia	2	1	number decreased dyserythropoiesis	number decreased dysmyelopoiesis
number decreased	leucopenia	2	number decreased	thrombocytopenia	2	2	number decreased dyserythropoiesis	number increased dysmyelopoiesis

normal in number n morphology	normal	0	normal in number n morphology	normal	0	1	micro normoblastic maturation dyserythropoiesis	normal maturation
number decreased	leucopenia	2	number reduced giant platelets	thrombocytopenia	2	1	number increased megaloblastic maturation dividing cells	megaloblastic maturation
number decreased	leucopenia	2	normal number n morphology	normal	0	1	number decreased megaloblastoid maturation	number decreased dysmyelopoiesis
normal number n morphology	Normal	0	normal number n morphology	normal	0	0	number increased megaloblastic maturation	normal number n maturation
normal number n morphology	Normal	0	normal number n morphology	normal	0	0	number increased megaloblastic maturation	normal number n maturation
normal number n morphology	Normal	0	normal number n morphology	normal	0	2	number increased megaloblastic maturation	number increased megaloblastic maturation
number increased shift to left	reactive neutrophilia	1	normal number n morphology	normal	0	2	number increased megaloblastic maturation	increased giant metamyelocytes band forms
normal number n morphology	Normal	0	normal number n morphology	normal	0	2	number increased megaloblastic maturation	number increased giant metamyelocytes
normal number n morphology	normal	0	normal number n morphology	normal	0	0	number normal megaloblastic maturation	number normal giant metamyelocytes band forms
normal in number n morphology	Normal	0	normal in number n morphology	normal	0	2	number increased megaloblastic maturation	number increased myelocyte metamyelocyte giant band forms

number decreased	leucopenia	2	number decreased normal morphology	thrombocytopenia	2	2	number increased megaloblastic maturation	number increased megaloblastic maturation
increase in neutrophils	RN	1	normal number and morphology	normal	0	2	number increased megaloblastic maturation	increase in metamyelocytes giant band forms
normal in number n morphology	normal	0	normal in number n morphology	normal	0	0	number increased megaloblastic maturation	megaloblastic maturation
normal in number n morphology	normal	0	normal in number n morphology	normal	0	2	number increased megaloblastic maturation	number increased dysmyelopoiesis
	normal	0		normal	0	0		
normal number n morphology	normal	0	normal number n morphology	normal	0	0	number increased normal maturation	number increased normal maturation
normal number n morphology	normal	0	normal number n morphology	normal	0	0	number normal normoblastic maturation	number increased normal maturation
increase in neutrophils toxic vacuoles n granules	RNL	1	number reduced normal morphology	thrombocytopenia	2	2	number normal micronormoblastic maturation	increase in metamyelocytes, band forms, mature neutrophils
normal in number n morphology	normal	0	number increased normal morphology	reactive thrombocytosis	1	2	number increased micro n macro normoblastic maturation	normal number n morphology
increase in neutrophils	reactive neutrophilia	1	normal in number n morphology	normal	0	0	number increased normal maturation	normal number n maturation
normal number n morphology	Normal	0	normal number n morphology	normal	0	0	number increased micro n macronormoblastic maturation	normal number n maturation

	normal	0		normal	0	0		
	normal	0		normal	0	0		
increase in neutrophils shift to left	reactive neutrophilic leucocytosis	1	normal number n morphology	normal	0	2	number normal early megaloblasti c maturation	increase in band forms
normal	normal	0		normal	0	0		
increase in neutrophils	reactive neutrophilia	1	normal number n morphology	normal	0	2	number increased micronormo blastic maturation	normal number n maturation
increase in neutrophils	RN	1	number increased normal in morphology	reactive thrombocyto sis	1	0	number increased micro n macro normoblastic maturation	incresed in number normal maturation
normal number n morphology	normal	0	reduced in number	thrombocyto penia	2	0	number increased micronormo blastic maturation	normal in number n morphology
increase in number shift to left	reactive neutrophilia	1	normal number n morphology	normal	0	0	number increased micronormo blastic maturation	number increased normal maturation
normal number n morphology	normal	0	normal number n morphology	normal	0	0	number increased micronormo blastic maturation	normal in number n maturation
increase in neutrophils	reactive neutrophilia	1	number decreased giant platelets	thrombocyto penia	2	0	number increased normal maturation	number increased normal maturation

normal number n morphology	normal	0	number increased giant platelets	reactive thrombocytosis	1	0	number increased micronormoblastic maturation	normal number n maturation
normal in number n morphology	Normal	0	normal in number n morphology	normal	0	2	number increased micro normoblastic maturation	normal in number n morphology
normal in number n morphology	RN	1	normal in number n morphology	normal	0	2	number increased micro n macro normoblasti maturation	normal in number n morphology
normal number n morphology	normal	0	normal number n morphology	normal	0	2	number increased, micro and macro normoblasti maturation	normal number n morphology
normal number n morphology	Normal	0	normal number n morphology	normal	0	2	number increased micronormoblastic maturation	normal number n maturation
normal number n morphology	normal	0	normal number n morphology	normal	0	2	number increased normal maturation	number increased normal maturation
normal in number n morphology	Normal	0	normal number n morphology	normal	0	0	number increased normal maturation	normal number n maturation
normal number n morphology	normal	0	number increased normal morphology	reactive thrombocytosis	1	0	number increased micro n macro normoblastic maturation	normal number n maturation
rphologynormal number n mo	normal	0	normal number n morphology	normal	0	0	number increased micronormoblastic maturation	normal number normal maturation
	normal	0		normal	0	2		

decreased	AML-M1	AML
normal in number n morphology	CML - accc. phase	CML
number increased normal morphology	CML - accelerated phase	CML
normal number n morphology	CML chronic phase	CML
	combined deficiency	combined
	combined deficiency	combined
	combined deficiency	combined
	combined deficiency	combined
	combined deficiency	combined
	combined deficiency	combined

	erythroid hyperplasia withmegaloblastic maturation consistent	
normal number n morphology	with megaloblastic anemia	
normal number n morphology	florid erythroid hyperplasia with micronormoblastic maturations/ o IDA	
	hyperplastic marrow with micronormoblastic maturation	
	hyperplastic marrow with micronormoblastic maturation	
	hyperplastic marrow with micronormoblastic maturation	
variable	MDS RCMD	MDS
normal morphology	MDS RCMD	MDS
normal number dysmorphic megks	MDS-RCMD	MDS
reduced number dysmorphic forms	MDS trilineage dysplasia - RAEB	MDS

dysmorphic megks	plasmacytosis	MDS with plasmacytosis	MDS
normal number n morphology		megaloblastic anemia	
normal		megaloblastic anemia	
normal number n morphology		megaloblastic anemia	
normal number n morphology		megaloblastic anemia	
normal number n morphology		megaloblastic anemia	
normal number n morphology		megaloblastic anemia	
normal number n morphology		megaloblastic anemia	
normal number n morphology		megaloblastic anemia	
normal in number n morphology		megaloblastic anemia	

normal
number n
morphology

megaloblasti
c anemia

normal in
number n
morphology

megaloblasti
c anemia

normal
number n
morphology

megaloblasti
c anemia

normal in
number n
morphology

megaloblasti
c marrow
with
dysmyelopoi
esis s/o MDS-
RA

MDS

micronormo
blastic
maturation

normal
number n
morphology

micronormo
blastic
maturation

normal
number n
morphology

micronormo
blastic
maturation

normal
number n
morphology

micronormo
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micronormo
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maturation

normal
number n
morphology

micronormo
blastic
maturation

micronormo
blastic
maturation
micronormo
blastic
maturation

normocellula
r marrow
with
normal in micronormo
number n blastic
morphology maturation
reactive
hyperplasia
micronormo
blastic
maturation

normal reactive
number n marrow s/o
morphology IDA

reactive
marrow with
micro n
macronormo
blastic
normal in maturation
number n s/o combined
morphology deficiency combined

Reactive
marrow with
normal in micro
number n normoblastic
morphology maturation

reactive
marrow with
normal micro
number n normoblastic
morphology maturation

reactive
marrow with
micro
normal normoblastic
number n maturation
morphology s/o IDA

reactive
marrow with
micro
normal normoblastic
number n maturation
morphology s/o IDA

		Reactive marrow with	
normal number n morphology		micronormo blastic maturation	
normal in number n morphology		REH s/o IDA	
normal in number n morphology		REH with micro n macro normoblastic maturation s/o combinrd deficiency	combined
normal number n morphology	plasmacytosis	REH with micro n macro normoblasti maturation and plasmacytosis	combined
normal number n morphology		REH with micro normoblastic maturation s/o IDA	
normal number n morphology		REH with micronormo blastic maturation	
normal number n morphology		REH with micronormo blastic maturation	
normal number n morphology		REH with micronormo blastic maturation	
normal number n morphology		REHs/o IDA	
		combined deficiency	

ID No	Name	Age	Gender	Presenting Complaints	Present Co-morbidities 1-DM 2-HT 3-Chronic Renal Dis 4-Chr Liver Dis 5-APD 6- Immune 7-Menstrual 8-Chr GI Bleeds 9-Spleni 10-TB 11-Thyroid 12-Cancer 13-GU Bleeding 14-Diarrhoea 15-Others	Dietary History 1-Vegan 2-Non Vegan	Habits 0-No 1-Alcohol 2-Smoking 3-Tea/Cofee excess intake	Palor 1-Sclrea 2-Tongue 3- Both
31. 15364	patrakali	66	F	exertional dyspnoea	4,11	2	0	2
38. 38421	guruvammal	60	F	exertional dyspnoea	1,2	2	2	2
43. 3904	velayutham	65	M	chestpain giddiness	4,11	2	0	2
6.15779	kosalai	65	F	fatigue	5, 8	2	3	1
35. 16214	navaneetham	71	F	exertional dyspnoea	2	1	0	3
29. 6858	gandhi	85	M	hematemesis	. .	1	. .	3
4.4798	esther	62	F	abdominal pain dyspnoea	1,5	2	. .	1

					15			
44.	3422	shanmugathar	65	F	exertional dyspnoea pedal edema		2	2	1
	60		73	F	pedal eema chest pain fatigue	2, 5, 6	1	0	3
	67	selvaraj	62	M	easy fatiguability loss of apetite	2, 15	. .	2,3	3
42.	14794	soundarajan	62	M	exertional dyspnoea palpitation	2, 3	2	. .	2
13.	43705	madasamy	70	M	pedal edema dyspnoea	15	1	0	3
18.	44867	mariappan	62	M	pedal edema facial puffiness	1, 10	2	2,3	1
47.	3490	jesumichel	65	M	pedal edema loss of weight	12	2	0	1
	1.5654	saraswathy	77	F	dyspnoea, pedal edema decreased urine output	1	2	0	2
37.	4632	rajeshwari	63	F	easy fatiguability loss of apetite	1,2,, 6	2	0	1
	64		61	F	fatigue chest pain giddiness	15	2	3	3

					3			
11. 60969	lakshmi	66	F	fatigue diarrh				
					, ,			
68		71	F	fatigue vomiti				
					. .			
22. 40029	veeramariamn	70	F	fever abdominal pain				
10.2039	mariyal	63	F	giddiness exertional dyspnoea	12, 14	2	3	1
65		74	F	giddiness, chestpain palpitation	12	2	1	1
14. 45911	madathi	65	F	hemoptysis, fever	8, 12, 14	2	2	1
39. 19917	ramani	67	F	loss of apetite	8	2	2	3
50. 22754	saroja	68	F	pedal edema facial puffiness	1			
48. 76231	subbulakshmi	61	F	vomiting feve	1,2,3	.	0	3
48. 76231	subbulakshmi	61	F	vomiting feve	15	2	3	1

					..			
	52		76	F		2	1, 3	2
	61	gnanam	69	F	15	2	2	1
14.	45911	madathi	65	F	2 hemoptysis, fever	2	1, 2, 3	2
	6.15779	kosalai	65	F	1 fatigue	2	0	2
45.	3398	sudalaimadan	61	M	1,2,, 6 abdominal pain malena	2	0	1
15.	43507	gurusamy	70	M	2,5, dyspnoea	2	. .	1
17.	42094	subbiah	60	M	dyspnoea decreased urine output 15	. .	2	1
	63		60	M	10, 15 exertional dyspnoea	2	3	3
24.	47881	paramasivan	62	M	2,3,6 exertional dyspnoea pedal edema	2	.?	1
25.	48220	murugan	63	M	1 fever	2	3	3
41.	31693	kannimalar	70	M	5 giddiness	2	3	1

					15			
	56		61	M	giddiness			
					1			
	57	sudalaimuthu	73	M	giddiness			
					. .			
28.	14794	sundar raj	69	M	giddiness			
					2			
46.	4954	krishnan	69	M	giddiness pedal edema	2	3	1
					..			
36.	42317	ganapathy	66	M	loss of apetite fatigue			
					15			
	8.4471	santhivinayag	65	M	ulcer leg	2	3	1
					..			
20.	46728	esakki	72	M	weakness liml	2	1, 3	2
					15			
	8.4471	santhivinayag	65	M	ulcer leg	2	2	1

36. 42317	ganapathy	66	M	loss of apetite fatigue	1,2,3	.	0	3
46. 4954	krishnan	69	M	giddiness pedal edema	10, 15	2	3	3
54		66	F	fatigue	2, 10	2	1	1
9.9578	bagavathy	66	F	fatigue	2, 3, 10	1	3	2
16. 61113	shanmugasun	62	M	cough palpitation pedal edema	15	2	3	1
3.1364	seetha	65	F	dyspnoea on exertion	3,8,10,15	2	3	3
7. 12487	subramanian	65	M	dyspnoea chestpain	1	. .	0	3
49. 50267	ponnammal	66	F	exertional dyspnoea palpitation	5 , 10	2	0	1
55		60	F	giddiness				
10.2039	mariyal	63	F	giddiness exertional dyspnoea	5, 8	. .	3	2
58	velu	67	F	giddiness loss of apetite fever	14	2	3	1

62		81	F	loss of apetite vomiting fatigue	2,4	2	2,3	1
21. 50914	esakkkiamma	66	F	mass abdome	…	2	,	2
2.6784	saraswathy	65	F	mass P/V urinary disturbances	1,2,4,6	1	0	2
19. 49305	arumugam	76	M	facial puffiness pedal edema				
33. 47768	muthukrishna	72	M	giddiness pedal edema	, ,			
23. 45982	srinivasan	74	M	giddiness swaying while walking	4			
30. 9806	ramar	67	M	paraparesis				
5. 1400	pathmanaban	61	M	pedal edema abdominal pain				

Lymphadenopathy 0-No 1-Isolated 2-Generalized	Spleen 0-No 1-Minimal 2-Moderate 3-Marked	Liver 0-No 1-Mild 2-Moderate 3-Marked	Pedal Edema 0-No 1-Yes	PR	BP	Resp Rate	Systems Describe Changes	Radiology Describe Changes	Endoscopy Describe
1	1	0	0	71	140/70	19	mild splenomegaly	fatty liver mild splenomegaly	congested GI
0	1	1	1	73	170/100	20	ESM, hepatosplenomegaly	mild cardiomegaly	congested GI
1	1	0	0	71	140/70	19	ld splenomega	fatty liver mild splenomegaly	congested GI
0	3	1	0	88	100/70	18	liver just palpable massivesplenomegaly	cardiomegaly splenomegaly dilated portal vein	esophageal varices severe PHT/ gastropathy
0	0	0	1	96	140/90	26	NAD	mild splenomegaly	gastric mucosal prolapse
0	0	0	0	70	11o /70	23	hemic murmur, thyromegaly	B/L MRD type I CKD	normal
0	0	0	1	76	140/70	16	b/l crepts	cardiomegaly mild pleural effusion minimal ascitis	normal

0	0	0	0	104	140/80	26	NAD	NAD	normal
1	1	0	1	76	110/80	16	ld splenomega	splenomegaly cardiomegaly	normal
0	0	0	0	84	130/70	20	NAD	cardiomegaly	normal
1	0	1	1	78	160/100	23	der hepatomeg	B/L medical renal disease type III RPD changes	normal
0	0	0	1	68	110/ 80	14	venous hum	NAD	normal
0	0	0	1	88	120/80	14	ESM	normal	normal
0	0	0	0	64	130/ 80	14	ca breast	NAD	normal
0	0	0	0	82	110/80	16	ca breast on treatment	NAD	not done
0	0	0	1	66	130/100	26	NAD	fatty liver	not done
0	0	0	1	78	140/80	23	b/l crepts	pneumonitic changes mild cardiomegaly	not done

									not done
									not done
									not done
0	0	0	0	84	120/ 80	18	ca breast	NAD	not done
0	0	0	0	68	14/ 80	18	/c/o ca alveolu	NAD	not done
1	0	0	0	62	140/60	19	/c/o ca stomac	NAD	not done
0	0	0	0	78	150/60	21	hemic murmur, haemorrhoids	NAD	not done
									not done
0	2	0	0	86	130/90	22	hemic murmur, splenomegaly	splenomegaly	not done
0	2	0	1	76	110/60	14	hemic murmur, B/L crepts, splenomegaly	old PT, Splenomegaly, increased renal cortical echoes	not done

0	2	0	1	92	150/80	14	moderate splenomegaly	splenomegaly	not done
1	2	0	0	70	130/80	20	moderate splenomegaly	hepatosplenomegaly	not done
0	0	0	1	80	150/80	18	spastic quadriparesis	non compressive myelopathy	not done
0	0	0	0	82	110/80	16	breast on treatn	NAD	not done
0	0	0	1	66	130/100	26	NAD	fatty liver	not done
0	0	0	0	68	130/90	24	hemic murmur	mild cardiomegaly	not done
0	0	0	1	88	120/70	22	NAD	cardiomegaly	not done
0	0	0	1	68	140/90	20	hemic murmur	old PT cardiomegaly	not done
1	0	0	1	93	140/70	22	NAD	mild splenomegaly	not done
0	0	0	0	78	120/70	18	NAD	NAD	not done
0	0	0	0	84	130/80	16	hemic murmur	NAD	not done

									not done
									not done
									not done
0	0	1	1	96	130/80	15	hemic murmur	cardiomegaly moderate splenomegaly	not done
									not done
0	2	0	1	76	110/60	14	hemic murmur, B/L crepts, splenomegaly	old PT, Splenomegaly, increased renal cortical echoes	not done
0	2	0	1	92	150/80	14	moderate splenomegaly	splenomegaly	not done
1	2	0	0	70	130/80	20	moderate splenomegaly	hepatosplenomegaly	not done

0	2	0	0	86	130/90	22	hemic murmur, splenomegaly	splenomegaly	not done
0	0	0	1	68	140/90	20	hemic murmur	old PT cardiomegaly	not done
0	0	0	1	110	130/80	22	systolic murmur	NAD	pale UGI muc
1	0	0	0	63	120/80	16	NAD	old PT B/L small kidneys	pale UGI muc
0	0	0	1	80	150/70	14	ystolic murmu	cardiomegaly B/L minimal PE	pale UGI muc
0	0	0	1	85	120/ 70	18	systolic murmur B/L crepts	old PT B/L medical renal disease type I RPD changes	pale upper GI
0	0	1	1	76	110/70	22	mild hepatomegaly, B/L crepts	NAD	pale upperGIr
0	0	2	1	84	110/80	14	epigatric tenderness hepatomegaly	old PT	
1	2	1	1	72	120/60	15	splenomegaly, tender hepatomegaly	splenomegaly, renal calculi	
1	0	2	1	90	120/80	16	hepatomegaly	hepatomegaly	

0	1	1	1	92	120/80	22	jaundice hepatomegaly	hepatosplenomegaly
0	1	1	1	94	130/80	16	jaundice hepatomegaly	splenomegaly calculous cholecystitis

TRBC x106cells/cu mm	TLC x103cells/cu mm	PLC x105cells/cu mm	HB gms/dl	HCT	MCV	MCH	MCHC	Retic C
1.19	11.4	1.73	8.1	24.9	116.9	32.7	31.3	0.5
2.19	4.3	1.35	6.3	20.3	92.7	28.8	31.1	1.2
1.19	11.4	1.73	8.1	24.9	116.9	32.7	31.3	< 0.5
2.22	4.2	0.41	6.3	18.4	93.6	18.4	22.3	1.2
3.47	5	2.98	5.3	22.3	64.3	15.3	23.8	1
2.1	3.9	2.12	5.6	24	76.4	21.6	28.8	1
1.01	5.8	2.92	6.8	16.6	65.3	15.8	24.2	0.5

3.2	5.8	90	10.4	33.7	97.4	32.9	33.8	1.5
1.78	4.7	3.96	7.4	23.5	77.2	19.3	27.4	0.5
3	4.5	4.22	7	24	66	18.7	22.6	1
2.68	4.5	2.31	8.2	24.7	99.6	30.6	30.8	0.5
1.96	7.8	2.3	5.9	18.8	78.2	20.5	25.7	0.5
2.89	3.7	1.93	9.1	31	107.3	31.5	20.4	1.2
2.91	5.4	3.15	11.1	26.8	97.5	24.3	31.7	0.5
3.11	4.8	2.45	7.6	22.3	84.7	24.5	30.2	0.5
2.39	4.3	2.69	6.6	22.1	94	23.8	27.4	1.4
1.87	9.2	1.48	5.7	19.4	76.9	21.5	27.3	0.6

	5.6	2.29	8.2		69.6			0.5
	2.9	1.13	5.7		62.9			1
	3.8	1.06	11.2		77.6			1
2.2	6.5	2.98	10.3	32.3	88.7	25.6	32.1	0.5
2.88	6.5	3.32	10.2	33.1	66.6	26.2	29.7	0.5
2.26	5.2	2.13	6.2	24.8	68.7	23.2	25.7	0.5
2.99	6.3	4.32	8.9	30.1	78.9	21.2	32.2	<0.5
	5.3	2.73	6.3		59.7			0.5
2.2	145	0.28	6.9	18.9	85.9	31.5	36.5	1
1.8	125	3.19	7.7	21.3	83.3	26.1	31.3	0.5

2.12	90,000	1.74	8.9	20.9	63.7	21.3	27.4	1
1.83	1.53	3.24	6.7	22.1	106	23.2	26.9	0.5
1.63	1200	1.27	7.7	25.6	104	31.3	30.1	0.6
3.11	4.8	2.45	7.6	22.3	84.7	24.5	30.2	1.5
2.39	4.3	2.69	6.6	22.1	94	23.8	27.4	1
3.3	12	3.3	9.1	36	89.4	23.6	30.1	1
2.63	5.9	2.94	9.1	29.6	100.4	22.6	28.3	0.3
1.56	3.2	0.9	6.8	19.6	128	18.7	26.6	0.5
1.92	2.9	71000	4.8	16	83.3	24.5	29.4	0.3
2.5	6.9	2.5	5.6	16	89.3	23.6	27.9	3
3	10.5	1.8	6.2	26	74	22	22	0.5

	5.3	2.84	10.8		90.4			0.5
	7.8	2.78	7.5		83.2			1
	4.7	3.16	9.3		87.4			1
1.95	7.3	1.42	5.1	13.8	70.8	16.4	23.2	0.5
	6.3	1.85	6.8		81.7			2
1.8	125	3.19	7.7	21.3	83.3	26.1	31.3	. .
2.12	90,000	1.74	8.9	20.9	63.7	21.3	27.4	. .
1.83	1.53	3.24	6.7	22.1	106	23.2	26.9	. .

2.2	145	0.28	6.9	18.9	85.9	31.5	36.5	. .
1.56	3.2	0.9	6.8	19.6	128	18.7	26.6	0.5
2.34	5.3	2.05	6.4	28.1	73.4	24.5	26.2	1.5
2.09	5.4	2.31	7.1	26.4	65.5	16.7	25.1	1.1
2.23	6.4	2.11	8.3	20.6	98.4	22.1	28.4	1.3
2.5	3.2	0.4	6.8	22.4	128	27	32	0.5
3.1	6.2	1.5	6.1	18	102.4	23.2	26.8	1
2.28	7.8	2.95	5.9	14.4	63.2	14.9	23.6	1.2
	4.1	1.52	7.7		86.6			1
2.7	11	0.19	6.4	19.3	72.5	21.3	31.1	0.5
1.96	10.6	3.55	5.8	20.1	66.4	21.2	29.6	0.5

2.99	7.3	2.65	8.3	20.2	56.5	19.3	22.4	0.5
	6.1	3.41	6.9		74.8			0.5
3.2	9.9	4.1	8.7	30.6	72.9	19.6	26.9	<0.5
1.88	6.5	3.26	8.3	36.8	86.5	22.6	28.7	0.5
	7.2	1.79	10.4		88.3			1
	7.4	2.53	8.7		82.6			2
	8.6	2.64	6.6		72.3			1.3
	9.1	1.02	7.9		86.6			0.5

ESR 1Hr	BSR	BUN	SCR	SBRT	SGOT	SGPT	ALP	SPROT
	74							
55 100	137	44	1	1.6	25	41	109	7
50, 105	96	129	2.2	1.2	36	26	206	5.3
32 60	98	63	1.2	2.7	105	123	197	5.8

110 150	106	29	0.8	0.9	32	21	96	6.2
20 130	157	150	5.4	1.1	46	39	114	4.8
15 30	60	44	1.2	0.8	17	24	89	5.4
42	60	48	1.3	0.7	28	33	89	5.6
50	76	28	0.6	0.9	18	21	87	5.4
60	97	20	0.9	0.7	19	22	94	6.1
50 100	110	28	0.8	.	.	.	.	.

100 130	114	44	1.2	2.4	19	22	85	5.3
28/50	128	30	1.1	2.1	21	28	90	4.6
100 150	125	22	1	0.6	31	31	85	7.9
. .	146	38	1.1	0.6	18	29	107	5.3
100	75	41	1.1	1.2	37	44	107	5.6
55 110	86	40	0.8	0.9	21	32	97	6.2
40	92	19	1	1.1	16	21	76	6.2
45, 85	65	41	1.2	3.8	48	24	65	6.7

10 40	90	21	0.8	0.8	24	21	108	6.2
40 85	89	32	0.9	0.8	34	28	116	6.1
50, 100	74	24	0.8	1	42	36	151	8.6
100 150	104	29	0.9	0.8	41	36	112	5.2
48	66	22	0.8	0.7	34	42	92	5.8
30 50	106	22	1.1	0.6	36	29	99	5.3
26 80	76	16	0.6	1.2	34	22	107	6.1
55	64	21	0.6	0.9	18	26	100	4.9

50 105	117	76	1.4	0.6	26	26	145	5.9
80 120	78	36	0.9	1.1	.	.	.	.
130	112	35	1.1	1.2	26	41	101	6.3
30 50	112	42	0.9	1.1	20	24	88	5.3
100 130	126	29	1.1	0.9	32	40	112	6
. .	68	20	0.8	.	.	.	.	.
98 130	70	30	1.1	0.7	20	22	102	4.6
60	73	38	1.2	0.5	62	66	110	4.9

.	92	65	3	. .	. .	..	. .	. .
40	92	19	1	1.1	16	21	76	6.2
110 150	106	29	0.8	0.9	32	21	96	6.2
10 22	137	27	0.7	2.4	63	54	109	4.8
100 132	132	95	4.3	0.6	40	20	104	7.1
..	113	43	0.6	..	.	.	.	.
100 130	65	50	1	1	38	27	79	6.3
110	102	36	0.9	1	26	28	88	6.2

52	35 3	28	1.3	0.6	18	22	86	5.8
50	94	23	0.9	0.6	34	36	102	5.4
5, 20	75	16	1.3	0.5	18	21	88	5.9
12, 45	178	56	1.5	0.6	46	37	99	7.5
55 120	148	24	0.8	1	40	36	104	5.7
30 60	146	21	0.8	1.1	41	44	118	4.8
10 22	115	34	1.5	0.4	34	42	98	6.1
55 100	146	20	0.8	0.9	40	36	106	5.2

ALB	T3	T4	TSH	PSS-RBC Describe RBC	PSS-RBC Impression	PSS-RBC 1- Normocytic 2-Microcytic 3-Macrocytic 4-Dimorphic 5-Others	PSS-WBC Describe	PSS-WBC Impression
					normocytic anemia	1		RN
					dimorphic anemia	4		RN
4		1.4	70	0.5	number decreased microcytic and macrocytic RBCs	MCHC anemia	2	normal in number n morphology	normal
				0.7		dimorphic anemia	4		normal
						MCHC anemia mild anisocytosis	2	n	RN
2.6		1.3	86	0.6	number decreased microcytic and macrocytic RBCs	dimorphic anemia	4	normal in number n morphology	normal
3		1.5	61	0.5	number decreased microcytic n normocytic	dimorphic anemia	4	normal in number n morphology	normal

3.2		68	4.5	2.1	number decreased normocytic n macrocytic RBCs	macrocytic anemia	3	increase in neutrophils	reactive neutrophilia
2.8	.	.	.		number decreased microcytic and macrocycytic RBCs	dimorphic anemia	4	increase in neutrophils	RN
2.3	.	.	.		number decreased normocytic normochromic RBCs occassional macrocyte	normocytic anemia	1	increase in neutrophils shift to left	reactive neutrophilic leucocytosis
2.8	.	.	.		number decreased microcytic n macrocytic RBCs	MCHC anemia	2	increase in neutrophils	reactive neutrophilia
2.5		1.3	91	0.4	number decreased microcytic hypochromic RBCs	MCHC anemia	2	normal in number n morphology	Normal
3.2		1.2	76	0.6	number decreased microcytic hypochromic RBCs	MCHC anemia	2	normal number n morphology	normal
.		1.2	65	0.7	number decreased microcytic hypochromic RBCs	MCHC anemia	2	rphologynormal number n mo	normal
						normocytic anemia	1		Normal
						MCHC anemia	2		RN
				1.3		MCHC anemia	2		normal

				0.4		normocytic anemia	1		normal
2.2		1.7	89	1	number decreased microcytic hypochromic RBCs	MCHC anemia	2	increase in neutrophils toxic vacuoles	reactive neutrophia
2.5	.	.	.		number decreased microcytic hypochromic RBCs	MCHC anemia	2	number decreased	leucopenia
4.4	.	.		1	number decreased microcytic hypochromic RBCs	MCHC anemia	2	number decreased	leucopenia
2.2		1.1	86	0.7	number decreased normocytic n macrocytic RBCs	macrocytic anemia	3	normal number n morphology	Normal
3.2	.	.	.		number decreased microcytic n macrocytic RBCs	dimorphic anemia	4	normal number n morphology	Normal
2.9	. .	. .		3.5	number decreased microcytic n macrocytic RBCs	dimorphic anemia	4	number increased shift to left	reactive neutrophilia
3.1	.	.		0.7	number decreased macrocytic RBCs	macrocytic anemia	3	number decreased	leucopenia
3.2		1.2	89	2.3	number decreased microcytic n macrocytic RBCs	dimorphic anemia	4	normal in number n morphology	normal
				0.4		MCHC anemia	2		RN

3.1		1.8	92	0.6	number decreased normoctic normochromic RBCs	normocytic anemia	1	normal number n morphology	normal
				0.8		MCHC anemia	2		normal
2.9	.	.	.		number decreased microcytic hypochromic RBCs	MCHC anemia anisopoikilocytosis	2	normal number n morphology	normal
4.6	.	.	.		number decreased normoctic normochromic RBCs	normocytic anemia	1	normal number n morphology	normal
2.9		0.5	74	0.8	number decreased microcytic hypochromic RBCs	MCHC anemia	2	increase in neutrophils toxic vacuoles n granules	RNL
2.7		1.1	83	0.7	number decreased microcytic hypochromic RBCs	MCHC anemia	2	normal number n morphology	Normal
				1.7		MCHC anemia	2		normal
				1.9		normocytic anemia	1	normal	normal
2.8	.	.	.		number decreased normocytic n microcytic RBCs anisopoikilocytosis	dimorphic anemia	4	increase in neutrophils	RN
3		1.3	91	0.4	number decreased microcytic hypochromic RBCs	dimorphic anemia	4	normal number n morphology	normal
2.7		0.9	91	0.3	number decreased microcytic hypochromic RBCs	MCHC anemia	2	increase in number shift to left	reactive neutrophilia

2.5					number decreased microcytic hypochromic RBCs	MCHC anemia	2	normal number n morphology	normal
.	.	.	.		number decreased normocytic normochromic RBCs	normocytic anemia	1	increase in neutrophils	reactive neutrophilia
3.2		1.5	61	0.5	number decreased microcytic hypochromic RBCs	MCHC anemia	2	normal number n morphology	normal
3.2					number decreased microcyti hypochromic RBCs	MCHC anemia	2	normal in number n morphology	Normal
2.9		1.2	69.1	0.6	number decreased microcytic n macrocytic RBCs	dimorphic anemia	4	number normal hypersegmented neutrophils	normal
. .		0	0	0	number decreased microcytic hypochromic RBCs	MCHC anemia NRBC	2	increase in mature neutrophil, band forms, myelocyt basophils	CML-accelerated phase
2.7	.	.	.		number decreased microcytic hypochromic RBCs	MCHC anemia	2	increased in neutrophils band forms occassional blasts	CML - chronic phase
2.2	.	.	.		number decreased macrocytic RBCs	macrocytic anemia	3	number increased mature neutrophils, band forms myelocytes occasssional blasts	CML - acc.phase

. .		0	0	0	number decreased microcytic hypochromic RBCs	MCHC anemia	2	increased myelolasts	acute leukemia
3.1	.	.		0.7	number decreased macrocytic RBCs	macrocytic anemia	3	number decreased	leucopenia
3.2		68	4.5	2.1	number decreased normocytic n macrocytic RBCs	macrocytic anemia	3	increase in neutrophils	reactive neutrophilia
3.1		0.5	78	0.9	number decreased schistocytes	hemolytic blood picture MCHC	2	normal number n morphology	RN
3	.	.		1.1	number decreased microcytic hypochromic RBCs	MCHC anemia	2	increase in neutrophils	reactive neutrophilia
.	.	.	.			MCHC anemia	2		Normal
				0.6		MCHC anemia	2		normal
				0.7		dimorphic anemia	4		normal
3.2		2	92	3.3	number decreased microcytic n normocytic RBCs	dimorphic anemia	4	number decreased	leucopenia
				1.3		MCHC anemia	2		RN
2.9		0.7	74	1.9	number decreased normocytic BBCs	normocytic anemia NRBC	1	normal number n morphology	Normal

3.1	1.1	83	0.7	number decreased macrocytic RBCs	macrocytic anemia	3	normal number n morphology	Normal
3	1.2	66	0.4	number decreased microcytic n macrocytic RBCs	dimorphic anemia	4	normal number n morphology	normal
2.7	1.3	78	0.3	number decreased macrocytic RBCs	macrocytic anemia	3	normal in number n morphology	Normal
3.5	0.6	60	0.5	number decreased microcytic n macrocytic RBCs	dimorphic anemia	4	normal in number n morphology	normal
3.1	1.6	82	1.2	number decreased microcytic hypochromic RBCs	MCHC anemia anisopoikilo cytosis	2	normal in number n morphology	RN
2.6	1.8	88	0.6	number decreased microcytic hypochromic RBCs	MCHC anemia	2	normal number n morphology	normal
3.1	1.2	83	0.5	number decreased microcytic hypochromic RBCs	MCHC anemia	2	normal number n morphology	Normal
3.2	0.8	88	1.2	number decreased microcytic hypochromic RBCs	MCHC anemia	2	normal number n morphology	normal

PSS-WBC 0-Normal 1-Reactive 2-Suspicious 3-Malignancy 4-Inconclusive	PSS-PL Describe	PSS-PL Impression	PSS-PL 0-Normal 1-High 2-Low 3-Giant Pl	Marrow Cellularity 0-Normal 1-Hypo 2-Hyper	Marrow Erythroid Describe	Marrow Myeloid Describe	Marrow Megk Describe	Marrow Others Describe
1	n	normal	0	0				
1		normal	0	2				
0	number increased normal morphology	reactive thrombocytosis	1	2	number increased micro n macro normoblastic maturation	normal number n morphology	normal number n morphology	
0		normal	0	2				
1		normal	0	0				
0	normal in number n morphology	normal	0	2	number increased megaloblastic maturation	number increased dysmyelopoiesis	normal in number n morphology	
0	normal in number n morphology	normal	0	1	micro normoblastic maturation dyserythropoiesis	normal maturation	dysmorphic megks	plasmacytosis

1	normal number n morphology	normal	0	0	number increased megaloblastic maturation	normal number normal maturation	normal number n morphology
1	normal number and morphology	normal	0	2	number increased megaloblastic maturation	increase in metamyelocytes giant band forms	normal in number n morphology
1	normal number n morphology	normal	0	2	number normal early megaloblastic maturation	increase in band forms	normal in number n morphology
1	normal number n morphology	normal	0	2	number increased micronormoblastic maturation	normal number n maturation	normal number n morphology
0	normal number n morphology	normal	0	0	number increased normal maturation	normal number n maturation	normal number n morphology
0	number increased normal morphology	reactive thrombocytosis	1	0	number increased micro n macro normoblastic maturation	normal number n maturation	normal number n morphology
0	normal number n morphology	normal	0	0	number increased micronormoblastic maturation	normal number normal maturation	normal number n morphology
0		normal	0	0			
1		normal	0	0			
0		reactive thron	1	0			

0		normal	0	0			
1	number reduced normal morphology	thrombocytopenia	2	2	dividing cells dyserythropoiesis	dysmyelopoiesis	normal morphology
2	number reduced normal morphology	thrombocytopenia	2	2	number decreased micro n macronormoblastic maturation dividing cells dyserythropoiesis	number increased myeloblasts promyelocytes mature forms	variable
2	number decreased	thrombocytopenia	2	2	number decreased dyserythropoiesis	number increased dysmyelopoiesis	reduced number dysmorphic forms
0	normal number n morphology	normal	0	0	number increased megaloblastic maturation	normal number n maturation	normal number n morphology
0	normal number n morphology	normal	0	2	number increased megaloblastic maturation	number increased megaloblastic maturation	normal number n morphology
1	normal number n morphology	normal	0	2	number increased megaloblastic maturation	increased giant metamyelocytes band forms	normal number n morphology
2	number decreased normal morphology	thrombocytopenia	2	2	number increased megaloblastic maturation	number increased megaloblastic maturation	normal number n morphology
0	normal in number n morphology	normal	0	0	number increased megaloblastic maturation	megaloblastic maturation	normal number n morphology
1		decreased	2	1			

0	normal number n morphology	normal	0	1	number increased megaloblastic maturation	dysmyelopoiesis	normal number n morphology
0		normal	0	0			
0	normal number n morphology	normal	0	0	number increased normal maturation	number increased normal maturation	normal number n morphology
0	normal number n morphology	normal	0	0	number normal normoblastic maturation	number increased normal maturation	normal number n morphology
1	number reduced normal morphology	thrombocytopenia	2	2	number normal micronormoblastic maturation	increase in metamyelocytes, band forms, mature neutrophils	normal number n morphology
0	normal number n morphology	normal	0	0	number increased micro n macronormoblastic maturation	normal number n maturation	normal number n morphology
0		normal	0	0			
0		normal	0	0			
1	number increased normal in morphology	reactive thrombocytosis	1	0	number increased micro n macro normoblastic maturation	incresed in number normal maturation	normal in number n morphology
0	reduced in number	thrombocytopenia	2	0	number increased micronormoblastic maturation	normal in number n morphology	normal in number n morphology
1	normal number n morphology	normal	0	0	number increased micronormoblastic maturation	number increased normal maturation	normal number n morphology

0	normal number n morphology	normal	0	0	number increased micronormo blastic maturation	normal in number n maturation	normal number n morphology
1	number decreased giant platelets	thrombocyto penia	2	0	number increased normal maturation	number increased normal maturation	normal number n morphology
0	number increased giant platelets	reactive thrombocyto sis	1	0	number increased micronormo blastic maturation	normal number n maturation	normal number n morphology
0	normal in number n morphology	normal	0	2	number increased micro normoblastic maturation	normal in number n morphology	normal in number n morphology
0	normal in number n morphology	normal	0	2	number increased micro n macronormo blastic maturation dividing cells	number increased dysmyelopoi esis	normal in number n morphology
3	number increased normal morphology	thrombocyto sis	1	2	number decreased micronormo blastic maturation	increase in myeloblasts promyelocyt es metamyelocy tes basophils	number increased normal morphology
3	number increased normal morphology	thrombocyto sis	1	2	number decreased micronormo blastic maturation	increase in myeloblasts promyelocyt es metamyelocy tes	normal number n morphology
3	number increased normal in morphology	reactive thrombocyto sis	1	2	number decreased micro n macronormo blastic maturation	increase in myeloblasts promyelocyt es myelocytes band forms mature neutrophils	normal in number n morphology

3	number reduced	thrombocytopenia	2	2	number decreased micronormoblastic maturation	increased in number myeloblasts >90%	decreased
2	number decreased normal morphology	thrombocytopenia	2	2	number increased megaloblastic maturation	number increased megaloblastic maturation	normal number n morphology
1	normal number n morphology	normal	0	0	number increased megaloblastic maturation	normal number normal maturation	normal number n morphology
1	number increased	reactive thrombocytosis	1	0	number increased micronormoblastic maturation	normal number n maturation	normal number n morphology
1	normal in number n morphology	normal	0	0	number increased normal maturation	normal number n maturation	normal number n morphology
0	n	normal	0	0			
0		normal	0	0			
0		normal	0	2			
2	number decreased normal morphology	thrombocytopenia	2	1	number decreased dyserythropoiesis	number decreased dysmyelopoiesis	normal number dysmorphic megks
1		normal	0	1			
0	normal number n morphology	normal	0	0	number increased megaloblastic maturation	normal number n maturation	normal number n morphology

0	normal number n morphology	normal	0	2	number increased megaloblastic maturation	number increased giant metamyelocytes	normal number n morphology	
0	normal number n morphology	normal	0	0	number normal megaloblastic maturation	number normal giant metamyelocytes band forms	normal number n morphology	
0	normal in number n morphology	normal	0	2	number increased megaloblastic maturation	number increased myelocyte metamyelocyte giant band forms	normal in number n morphology	
0	number increased giant platelets	reactive thrombocytosis	1	2	number increased micro n macro normoblastic maturation dividing cells	normal in number n morphology	normal number n morphology	
1	normal in number n morphology	normal	0	2	number increased micro n macro normoblastic maturation	normal in number n morphology	normal in number n morphology	
0	normal number n morphology	normal	0	2	number increased, micro and macro normoblastic maturation	normal number n morphology	normal number n morphology	plasmacytosis
0	normal number n morphology	normal	0	2	number increased micronormoblastic maturation	normal number n maturation	normal number n morphology	
0	normal number n morphology	normal	0	2	number increased normal maturation	number increased normal maturation	normal number n morphology	

Marrow Impression Describe	diagnosis
combined deficiency	combined
combined deficiency	combined
micronormoblastic maturation	
hyperplastic marrow with micronormoblastic maturation	
combined deficiency	combined
megaloblastic marrow with dysmyelopoiesis s/o MDS-RA	MDS
MDS with plasmacytosis	MDS

erythroid hyperplasia withmegalob lastic maturation consistent with megaloblasti c anemia

megaloblasti c anemia meg ane

normocellula r marrow with micronormo blastic maturation

reactive marrow s/o IDA

REH with micronormo blastic maturation

REH with micronormo blastic maturation

REHs/o IDA

combined deficiency combined

combined deficiency combined

hyperplastic marrow with micronormo blastic maturation

hyperplastic marrow with micronormo blastic maturation	
MDS RCMD	MDS
MDS RCMD	MDS
MDS trilineage dysplasia - RAEB	MDS
megaloblasti c anemia	meg ane
megaloblasti c anemia	meg ane
megaloblasti c anemia	meg ane
megaloblasti c anemia	meg ane
megaloblasti c anemia	meg ane
megaloblasti c dysmegakary opoiesis	meg.mega

megaloblasti
c p/o MDS
ysmyelopoie meg
sis dysmyelo
micronormo
blastic
maturation

micronormo
blastic
maturation

micronormo
blastic
maturation

micronormo
blastic
maturation

micronormo
blastic
maturation

micronormo
blastic
maturation

micronormo
blastic
maturation
reactive
hyperplasia
micronormo
blastic
maturation

reactive
marrow with
micro n
macronormo
blastic
maturation
s/o combined
deficiency combined

Reactive
marrow with
micro
normoblastic
maturation

reactive
marrow with
micro
normoblastic
maturation

reactive marrow with micro normoblastic maturation s/o IDA	
reactive marrow with micro normoblastic maturation s/o IDA	
Reactive marrow with micronormo blastic maturation	
REH s/o IDA	
REH with micro n macro normoblasti maturation with features s/o myelodyspla sia	p/o MDS
CML - accelerated phase	CML
CML chronic phase	CML
CML - accc. phase	CML

AML-M1	AML
megaloblastic anemia	meg ane
erythroid hyperplasia withmegaloblastic maturation consistent with megaloblastic anemia	meg ane
florid erythroid hyperplasia with micronormoblastic maturations/o IDA	
micronormoblastic maturation	
combined deficiency	combined
micronormoblastic maturation	
combined deficiency	
MDS-RCMD	MDS
meg. Yserythropoiesis	meg. Yserythropoiesis
megaloblastic anemia	meg ane

megaloblastic anemia	meg ane
megaloblastic anemia	meg ane
megaloblastic anemia	meg ane
reactive marrow with micro n macronormoblastic maturationpossibility of MDSmay be thought of	p/o MDS dyserythropoiesis
REH with micro n macro normoblastic maturation s/o combinrd deficiency	combined
REH with micro n macro normoblasti maturation and plasmacytosis	combined
REH with micro normoblastic maturation s/o IDA	
REH with micronormoblastic maturation	